THE PHYSIOLOGY, HISTORY AND CONCEPTS

of

ELECTROCARDIOGRAPHY and VECTORCARDIOGRAPHY

by

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The author would appreciate notification of any errors.

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Preface

With the exception of this preface, which replaces the Purpose and Scope section, this paper constitutes the introduction of a Master of Science thesis, A VECTOR ELECTROCARDIOSCOPE SYSTEM FOR CLINICAL STUDIES, submitted to the Department of Electrical Engineering at Oregon State University in the summer of 1964. It was the intent of the thesis to acquaint a non-medically oriented, but practicing, electronics engineer with details of a system to be utilized as a research tool in the study of the heart and of heart disease. In order to have a certain amount of perspective and to assimilate the significance of the system, the author feels that the engineer must know something of the heart, of its terminology, of the concepts used in its study, and of the instruments used in its investigation. This thesis introduction should give such background information in sufficient depth to be truly meaningful. As such, it is being distributed as a reference document pertaining to electrocardiography and vectorcardiography.

The Heart, Its Task and Significance

Although the importance of the heart in the maintenance of life must clearly have been known by the ancients, it was not until 1616 that there was recorded a possible function for this vital organ. That

year William Harvey, an English physician, made an annotation in the Prelectiones Anatomiae Universalis, his 1616 lecture notes:

> WH constat per fabricam cordis sanquinem per pulmones in Aortam perpetuo transferri, as by two clacks of a water bellows to rayse water constat per ligaturam transitum sanquinis ab arterijs ad venas vnde perpetum sanguinis motum in circulo fieri pulsu cordis An? hoc gratia Nutrionis an magis Conservationis sanguinis et Membrorum per Infusionem calidam vicissimque sanguinis Calefaciens membra frigifactum a Corde Calefit

These rough notes may be freely translated as (49,p.xv):

> WH demonstrates by the structure of the heart that blood is continually passed through the lungs into the aorta, as by two clacks of a water bellows to raise water. The passage of blood from arteries to veins is shown by means of a ligature. So it is proved that a continual movement of the blood in a circle is caused by the beat of the heart. Is this for the sake of nourishing or the better preservation of the blood and parts of the body by infusion of heat, the blood being cooled, by heating these parts, and warmed, by the heart?

Harvey followed this first glimmer of heart function with a series of investigations over a period of eight years, all the while demonstrating his findings to a most critical audience (his colleagues). His scientific analysis, from a philosophical view, is one of the finest examples of deductive reasoning. In 1628 the results of his cardiovascular researches were published as the universally acknowledged masterpiece De Motu Cordis (49). a permanent historical milestone and one of the really great contributions to medicine.

The function of the heart is to pump blood. The importance of this task is manifest upon considering the functions of the blood (119, p.490): 1) transport of food, waste products, gases (O_2 and CO_2), and hormones; 2) regulation of body pH, fluid balance, and temperature;

3) defense against infection via phagocytic leukocytes and antibodies; and 4) self preservation through the prevention of hemorrhage. To its appointed task the heart brings a remarkable perseverance considering the staggering facts that in an average life time the heart beats approximately 2.5 billion times, pumps over 300 million liters (80 million gallons) of blood, and converts 2.5 billion joules (.7 million kilowatt hours) into hydraulic energy.

With all this work and no vacation it is not surprising that the heart tires and precipitates various degrees of disability. It is estimated that about ten million Americans suffer from heart or cardiovascular related diseases (99,p.1), which are a major health problem at all age levels. These diseases are the cause of death of more than 900,000 Americans annually; they cause at least one out of every two deaths in the nation. The statistics for the year 1961 are (2,p.6): 928,670 deaths, approximately three and one-half times the number of deaths due to cancer. In Oregon heart diseases are the #1 health problem (98,p.1), being annually responsible for about 56% (9,400 persons) of all deaths in the state. One person in Oregon dies every 45 minutes from cardiovascular disease. There are over 150,000 Oregonians suffering from diseases of the cardiovascular system, including 7,500 children.

With the significance of heart disease in mind it is easily understood that anything which aids in the understanding of the physiology of the heart, the pathology of heart disease, and the correct diagnosis of cardiac disorders is of immense importance. One such device is the electrocardiogram, commonly called the ECG, or from the German elektro-

kardiogram, the EKG.

Basic Electrocardiography

Physiology of the Heart

Mechanical activity

The heart is a bicameral pump which in the average resting man supplies approximately 80 ml of blood to the body (systemic circulation) and 80 ml of blood to the lungs (pulmonary circulation) with each heart beat (39,p.421). This blood volume is forced into the systemic circulation at a pressure of approximately 100 mm Hg. and into the pulmonary circulation at approximately 15 mm Hg. This hydraulic energy results from the contractile forces of the heart muscle, the myocardium. The myocardium, by simultaneously shortening and compressing the inner chambers of the heart squeezes the blood into the systemic and pulmonary circulations, which have substantial heads of back pressure.

The cardiac cycle is characterized by the following mechanical events. Between beats the heart mechanically rests, and this is known as the period of diastole (Gr. - dilatation). During diastole the heart passively fills with oxygenated blood returning from the lungs and oxygen-poor venous blood returning from the body. The pulmonary blood passes into the left atrium (L. - entrance hall), through the left atrioventricular (bicuspid or mitral) valve, and on into the left ventricle (L. - belly). The systemic blood passes into the right atrium, through the right atrioventricular (tricuspid) valve, and on into the outports by the semilunar valves, which are held closed by the back

pressure in the systemic and pulmonary circulations.

The heart's period of mechanical activity is known as systole (Gr. - contraction). The onset of systole is initiated by the contraction of the atria which propels additional blood (approximately 40%) into the ventricles. Within two-tenths of a second (4,p.341) the ventricles begin to contract and thereby cause a rise in ventricular pressure. This increased pressure shuts the atrioventricular valves, and with further contraction the pressure continues to rise. Since all the valves are shut, the ventricular volumes remain constant, and this is known as the period of isometric contraction. Once the pressure of the systemic and pulmonary circulations are exceeded, a phase of ventricular ejection is begun. The left semilunar (aortic) valve is forced open and blood is squeezed into the systemic circulation. Likewise the right semilunar (pulmonary) valve supplies blood to the pulmonary circulation. After the ventricular contents are ejected, the myocardium relaxes and the ventricular pressures fall. As soon as these pressures fall below the pressures sustained in the circulatory systems, the semilunar valves close. This signals the onset of diastole, and until the ventricular pressures are low enough for the atrioventricular valves to open, there is a phase of isometric ventricular relaxation. The mechanical events of the cardiac cycle are illustrated in Figure 1 (39,p.416).

The mechanical activity of the heart is brought about by the contractile forces within the striated cardiac muscle fibers that make up the myocardium. Striated cardiac fibers are in many respects similar, but not identical, to the striated skeletal muscle fibers that make up

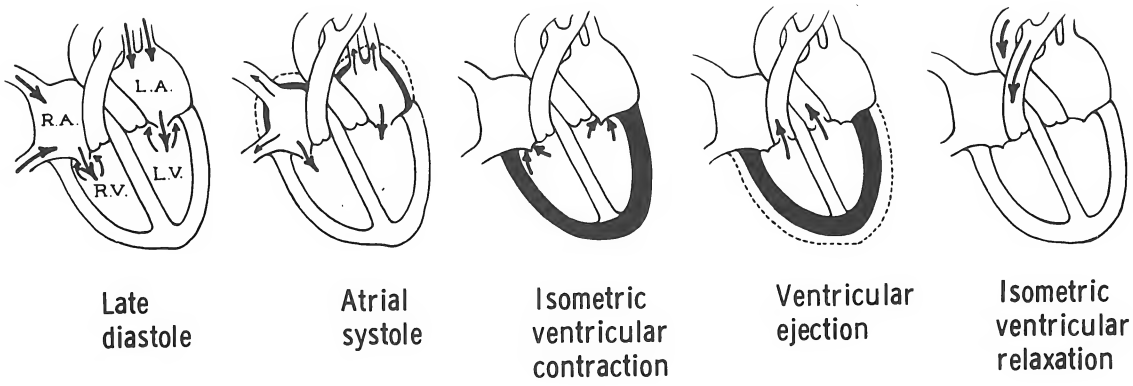


Figure 1. Blood flow in the heart and great vessels during the cardiac cycle. The portions of the heart contracting in each phase are indicated in black. RA and LA, right and left atria; RV and LV, right and left ventricles.

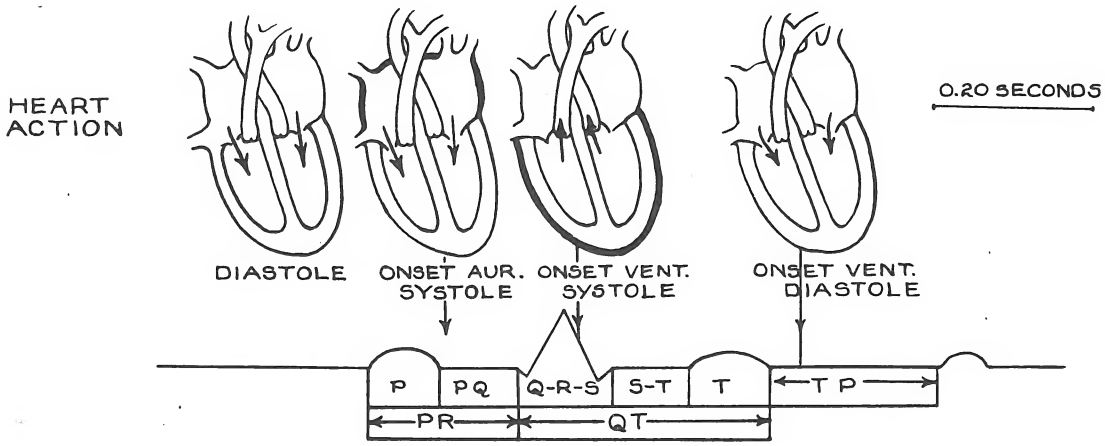


Figure 2. Relation between electrical and mechanical events in the cardiac cycle. The passage of the electric impulse through any given portion of the heart precedes the resulting contraction of that portion of the myocardium. The vertical arrows indicate the timing of the mechanical events. The bottom drawing is a schematized ECG indicating the timing of the electrical events.

the external muscles of the human body. It is currently held (66,p.133) that for skeletal muscle the sliding filament model advanced by Huxley in 1954 (65,p.255) is still strongly supported by the bulk of the evidence today. Briefly, the theory suggests that the parallel protein fibrils which are longitudinally oriented within the muscle fiber are interdigitated with each other, much like the control and fuel rods of a nuclear reactor. During contraction these filaments haul themselves along each other through the interaction of spatially organized chemically active sites located along the filament surfaces. Now the mechanisms of cardiac contraction may well be different, in fact there are some observations of the myocardium that tend to favor a protein folding theory over the purely sliding filament model (6,p.155). Today the similarities and differences between cardiac and skeletal muscle are under widespread and vigorous investigation. In fact over one half of all the cardiovascular research recorded in the medical literature is dated after the year 1955 (2,p.6). However, cardiac and skeletal muscle do have in common the fact that they are both preceded by electrical activity. This is illustrated in Figure 2 (46,p.39).

Electrical activity

It is almost universally accepted (9,p.341) that the electrical activity of the cardiac cell membrane initiates the development of tension. This is the case in skeletal muscle where it is well established that electrical activity of the surface membrane is the direct or indirect activator of contraction (131,p.101). Again, since the picture

is still not clear in the case of cardiac muscle, it is necessary to turn to the story for skeletal muscle. Electrical activity sweeping along the surface of a membrane is known as the membrane action potential. In the case of skeletal muscle the passage of this action potential releases Ca^{++} ions to the inner structure of the muscle fiber (66, p.141). These ions then trigger the contractile mechanism to develop tension. The results of current investigations have a trend which would seem to indicate that substantially the same story holds for cardiac muscle (9,p.353). However there are certain exceptions (9,p.349), and there is much work to be done on the correlation of cardiac action potentials and contractile activity.

Genesis of the Action Potential

Animal electricity has been the subject of continued investigation since 1731 when an Englishman Stephen Gray suspended a boy from a high ceiling, electrified him with a glass rod and friction, and then detected the electrification with an electroscope (11,p.15). A French version of this same experiment is illustrated in Figure 3 (11,p.16). The boy is being charged with a friction machine, and this charge is being passed onto a girl who is functioning as an electrometer by attracting with her other hand pieces of lint. In 1773 another Englishman John Walsh was making investigations into the intrinsic electricity of several animal forms, especially the marine torpedo and the electric eel (11,p.17). Aloisio Galvani, an Italian, published in 1791 his Commentarius (38) in which were described experiments on the nerve muscle

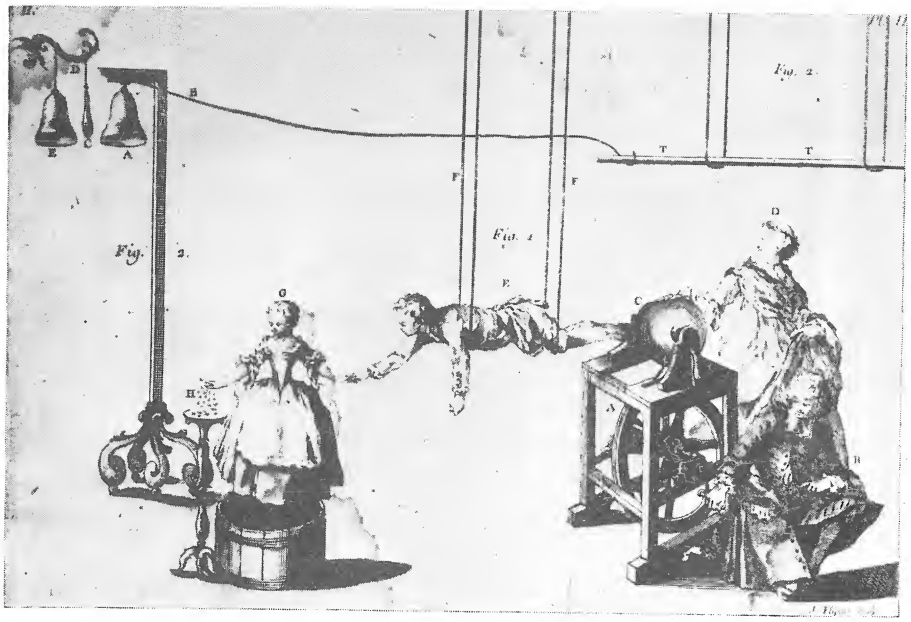


Figure 3. The experiment of electrifying a boy. From F. H. Winckler's Essai sur la Nature. Les effets et les causes avec description de deux nouvelles machines a Electricite. Paris, Jorry, 1748.

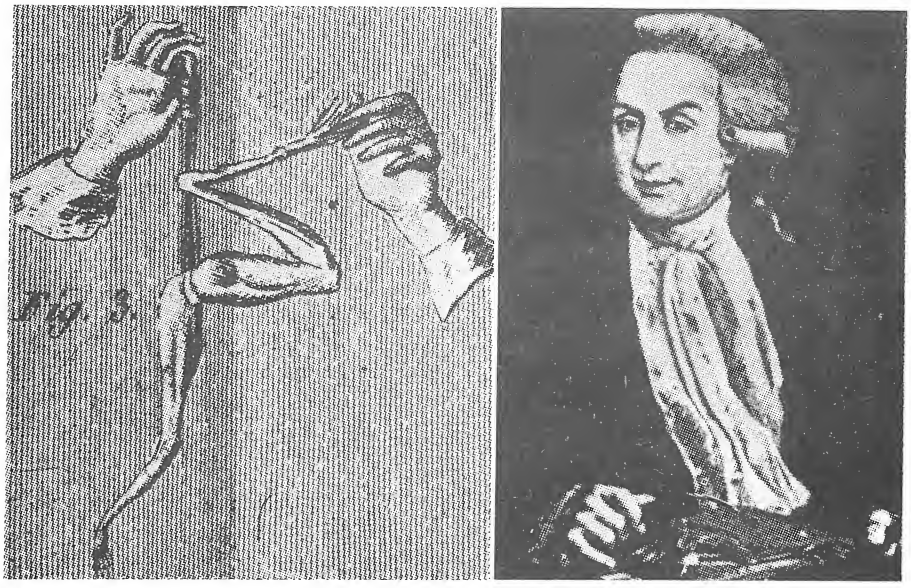


Figure 4. The nerve muscle preparation as used by Galvani to demonstrate intrinsic animal electricity (by muscle contraction) in the absence of bimetallic currents. The portrait is from a contemporary painting in the Library of the Univ. of Bologna.

preparation of the frog. Figure 4 shows such a preparation. He attempted to fully explain his results in terms of animal electricity, a view vehemently rejected by Alessandro Volta who was pushing, to tortuous lengths, the concept of bimetallic current. As pointed out by Frederick von Humboldt in 1797 (11,p.18) both men were somewhat right and somewhat wrong, and that Galvani had confused the discovery of two genuine, but not mutually exclusive, phenomena: intrinsic animal electricity and bimetallic electricity.

Towards the middle of the 19th century Matteucci, the Professor of Physics at Pisa, laid much of the ground work for muscle electrophysiology. Among his many contributions was the demonstration that the intrinsic electricity of the nerve-muscle preparation could exist in the muscle alone (11,p.20). Following this, du Bois-Reymond, a Swiss in Berlin, discovered the action potential of nerve with the following claim: "If I do not greatly deceive myself, I have succeeded in realizing in full actuality (albeit under a slightly different aspect) the hundred years' dream of physicists and physiologists, to wit, the identity of the nervous principle with electricity" (11,p.22). He also conceived of a scheme of regularly oriented electromotive particles arranged along the surface of muscle and nerve. This was the forerunner of a concept of polarization to be more accurately and fully developed later by his pupil Bernstein. Bernstein believed that due to the semipermeable nature of its membrane that the inactive muscle fiber was normally polarized, having positive ions on the inside. He hypothesized that the action potential was a self-propagating depolarization of

this membrane due to a breakdown of its semipermeable character (8). This is at the core of the modern day theory, and much of modern progress has been the establishment of experimental proof for this earlier theory.

Through energy dependent cellular metabolism there is an active transport of the Na^+ ion across the cell membrane from the interior of all cells to the interstitial fluid between cells (130,p.18). This efflux of Na^+ ions is countered by an influx of K^+ ions in order that electroneutrality may be approximately maintained. The result is that there is a wide disparity of ionic concentrations between the cellular contents inside and the interstitial fluid outside: Na^+ has a high concentration on the outside, while K^+ is high on the inside. Consider the well known form of the Nernst equation for univalent ions (130,p.16)

$$E = \frac{R\,T}{F} \ln \frac{(\text{Concentration})_{\text{outside}}}{(\text{Concentration})_{\text{inside}}}$$

$$= 60 \log_{10} \frac{(\text{ion})_o}{(\text{ion})_i} \text{ millivolts at } 37^\circ \text{ C.}$$

For the known concentrations in certain cases of both potassium and chloride this equation yields a potential of approximately -95 mv (negative inside of the cell), which is very close to the measured value of -90 mv (130,p.17). Now the concentration ratio of sodium is the reverse of that of potassium, and application of the Nernst equation gives +45 mv for the equilibrium potential. However, things are not in equilibrium for sodium, since there is a sodium pump which is actively transporting sodium out of the cell in order to maintain the

disequilibrium. Thus the membrane in a sense appears to be selectively permeable to potassium and chloride, but not permeable to sodium. An equilibrium potential equation that takes into account the mobility of ions was derived by Planck in 1890 (102): It is very complicated, but for the single-salt univalent case reduces to:

$$E = \frac{u - v}{u + v} \frac{RT}{F} \ln \frac{C_i}{C_o}$$

where u and v are anion and cation mobilities and C_i and C_o are the inside and outside concentrations.

A much simpler equation for the many-salt systems was derived by Goldman in 1943 (45). This equation has the refinement of using permeability coefficients P rather than mobilities, and thus accounts for the characteristics of membrane thicknesses, partition coefficients, and mobilities. As used by Hodgkin and Katz (60) the equation is:

$$E = \frac{RT}{F} \ln \frac{P_K(K)_i + P_{Na}(Na)_i + P_{Cl}(Cl)_o}{P_K(K)_o + P_{Na}(Na)_o + P_{Cl}(Cl)_i}$$

As applied to a cell membrane of the squid the equation yields for the resting membrane potential -60 mv, which is very close to the experimental value of -62 mv (60). For the same values of concentrations the Nernst equation yields the following equilibrium potentials: E_K = -89 mv: E_{Na} = +46 mv; and E_{Cl} = -55 mv. The positive influence of sodium has little effect on the calculated resting membrane potential in the Goldman-Hodgkin-Katz equation because its permeability coefficient P_{Na} is so much lower than that of both potassium and chloride. It should

be pointed out that the permeability coefficients are by no means constant. They vary with a large number of parameters: temperature, time, membrane potential, concentrations, gas tensions, etc.

From the time of du Bois-Reymond it was realized that during continuous muscle contraction (tetanus) there was a change in electrical activity, which he named the "negative variation" (11,p.22). His pupil Bernstein made the identification in nerve of this "negative variation" with the action potential (11,p.23). He also noted that at times the amplitude of the negative variation exceeded the value of the resting nerve potential; i.e., the deflection of his galvanometer sometimes crossed the base line, a portentous observation. A giant step forward was made possible in 1936 with the discovery of the giant (almost 1 mm in diameter) axon (the long conducting member of a nerve cell) of the squid Loligo forbesi (133,p.323). This allowed Hodgkin and Huxley in 1939 (54) to insert a micro-electrode (.1 mm in diameter) into the interior of a nerve cell. Thereby they directly measured the transmembrane potential of a living nerve membrane. Figure 5 (54,p.710) illustrates the micro-electrode within the axon. They measured a resting membrane potential of about -50 mv, and an action potential amplitude of about +90 mv. Figure 6 (54,p.711) is a photographic record of the transmembrane action potential. It is important to note that during the action potential the polarity across the membrane actually reverses (to +40 mV), a result not handled by the Bernstein hypothesis.

The currently accepted explanation was finally given by Hodgkin and Katz in 1949 (60). Their sodium ion hypothesis for the genesis

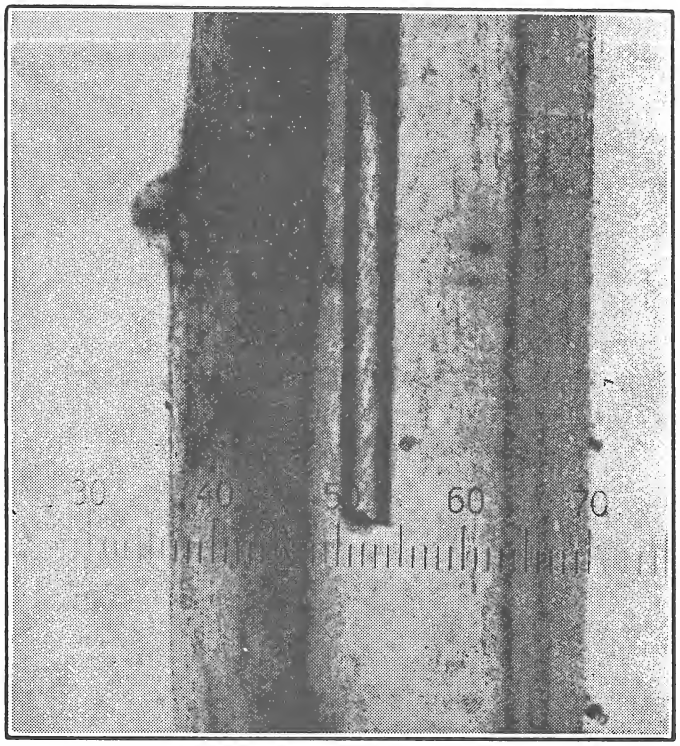


Figure 5. Photomicrograph of an electrode inside of a giant axon. Each scale division = 33 μ. The giant axon is the clear space between scale divisions 48 and 63, and is surrounded by smaller fibers and connective tissue. The glass microelectrode extends upward from the scale markings and contains at the top edge of the photograph a fine silver wire.

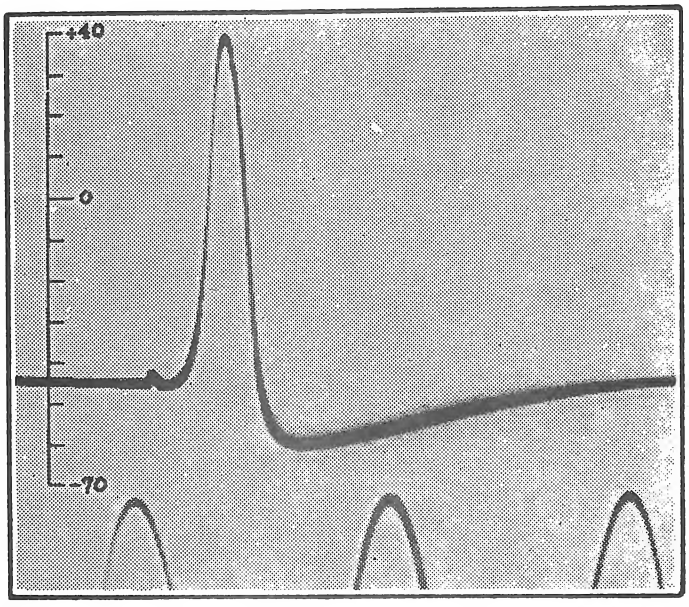


Figure 6. Action potential recorded between the inside and outside of a squid giant axon. Time markers, 500 cps. The vertical scale indicates the potential of the internal electrode (in millivolts) with reference to the sea water external to the axon.

of the action potential is supported by a considerable amount of evidence. In brief they postulated that the action potential was the result of a temporary 500 fold increase in P_{Na}, the membrane permeability to sodium. As a result of this, the influence of the sodium concentrations become dominant in the Goldman-Hodgkin=Katz equation. Therefore the transmembrane potential during the height of the action potential approaches the equilibrium potential of sodium as given by the Nernst equation.

In a series of five papers (55,56,57,58,59) Hodgkin, Huxley, and Katz in the early 1950's made a thorough study of the voltage-current relationships of the giant axon of Loligo. From this they were able to deduce empirical formulas for the variations of both sodium and potassium permeabilities (P_{Na} and P_K) with membrane potential and time. Using these equations they were then able to calculate the permeability changes taking place curing the time course of membrane electrical activity. Thereby they were able to mathematically reconstruct action potentials that agreed well with experiment. Figure 7 (58,p.530) illustrates a reconstructed action potential V and the associated conductances g_{Na} and g_K (which are respectively functions of the permeabilities P_{Na} and P_K). The conductance $g = g_{Na} + g_K$. Figure 8 (58,p.525) illustrates the rather remarkable similarity of the mathematical reconstructions of the action potential (above) and the actually recorded action potentials (below) for several different experimental conditions.

A significant outcome of the investigations was the representation

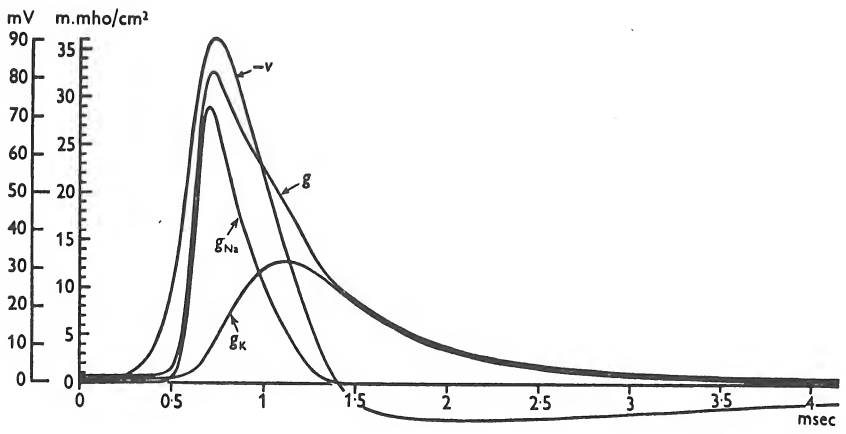


Figure 7. Reconstructed propagated action potential of the squid giant axon at 18.5° C., with values of the membrane conductances ($g = g_{Na} + g_K$). Transmembrane potential at rest is plotted as zero while action potential changes (outside minus inside) are plotted as -V with negative upward.

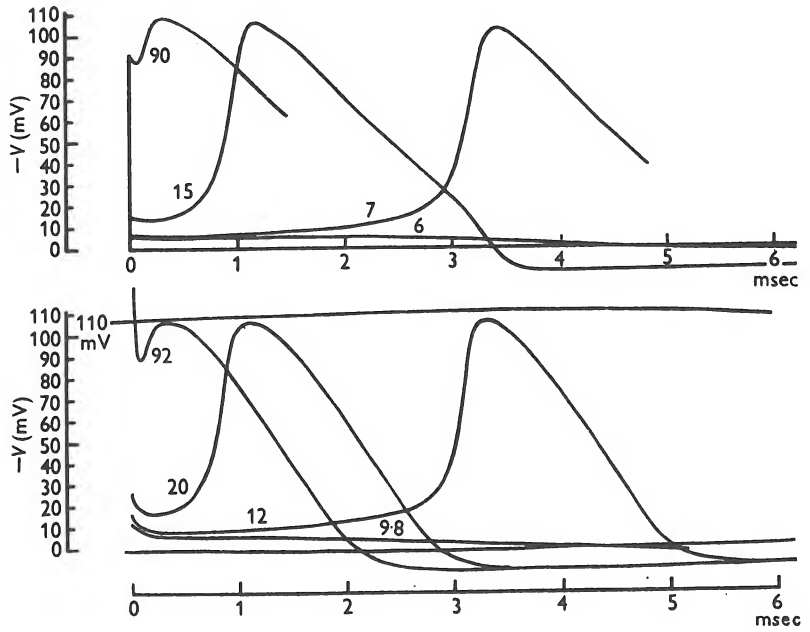


Figure 8. Upper family of curves; plots of the mathematical reconstructions of membrane potentials for initial depolarizations of 90, 15, 7, and 6 mV. Lower family: tracings of membrane action potentials recorded at 6° C. The attached numbers are the stimulus shock strength in μcoulomb/cm^2. Depolarizations (or negative displacements of V) are all plotted upwards.

of the squid giant axon membrane by the equivalent circuit as shown in Figure 9 (58,p.501). C_M represents the membrane capacitance: E, the membrane potential E_{Na}, the Nernst equilibrium potential for sodium; E_K, the Nernst equilibrium potential for potassium; E_1, the leakage potential made up from chloride and other ions; R_{Na}, the time and membrane potential dependent equivalent source resistance for the sodium battery; R_K, the time and membrane potential dependent potassium resistance; R_1, the fixed and rather large leakage resistance; I, the externally applied current, and I_{Na}, I_K, I_1 are the currents flowing in the respective branches. For the resting membrane the transmembrane potential is somewhere between E_K and E_1 as determined by the voltage divider R_K and R_1; R_{Na} is very high and therefore E_{Na} has little influence. However upon activation R_{Na} becomes very low, and therefore the transmembrane potential assumes a value between E_{Na} and E_K, even to the extent of reversing the transmembrane polarity. C_M is required in order to give the transition phenomena the proper time constant.

But what initiates the action potential? In Figure 6 or even more strikingly in Figure 8 there is noted a sharp increase in the slope of the action potential once it has increased approximately 15 mV above the resting level (which is -45 in Figure 6 and 0 in Figure 8). At this point (the threshold level) the influence of the membrane potential on R_{Na} becomes regenerative. Therefore R_{Na} decreases rapidly and there is a switchover in potential in the direction of the Nernst equilibrium potential for sodium. With the passage of time R_{Na} is again increased

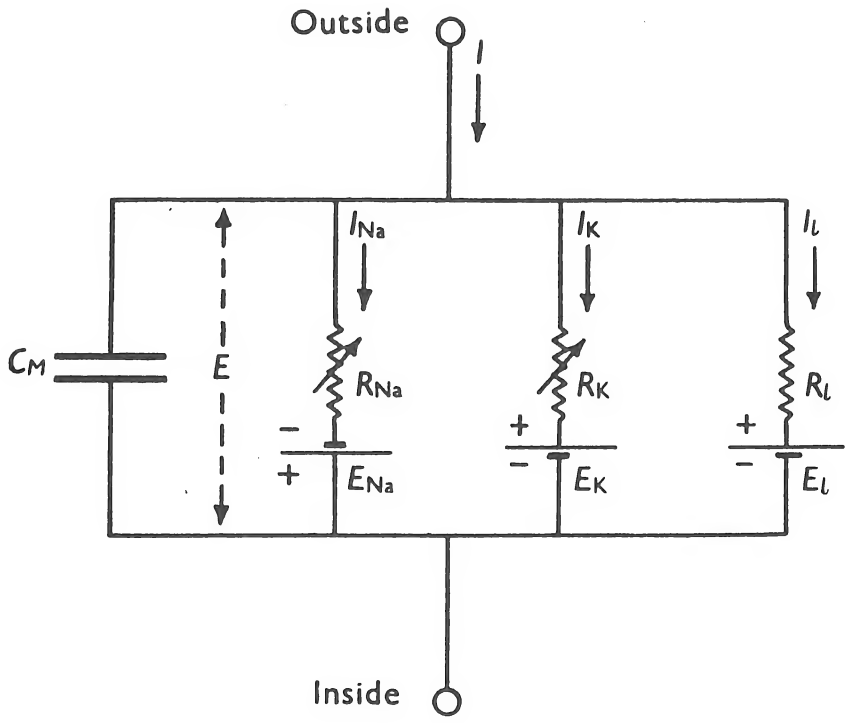


Figure 9. Equivalent circuit of the squid giant axon membrane. $R_{Na} = 1/g_{Na}$; $R_K = 1/g_K$; $R_1 = 1/g_1$. R_{Na} and R_K vary with time and membrane potential; the other components are constant.

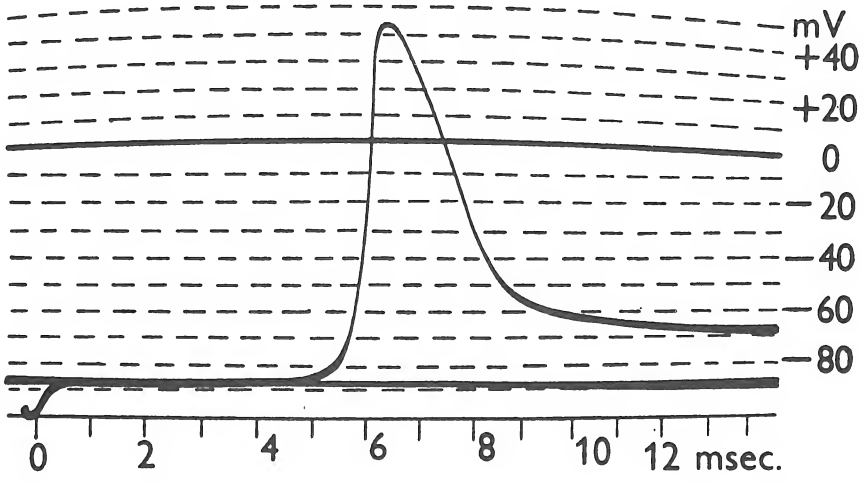


Figure 10. Action potential of the frog sartorius muscle fiber, recorded internally with a very fine micropipette. Resting potential, -88 mV. At peak of action potential, +43 mV. Temp, 13° C.

(see Figure 7) and the potential returns to the resting level, and the influence of potential on the membrane permeabilities is momentarily blocked (the nerve is said to be refractory). Thus anything that can raise the transmembrane potential of a nonrefractory nerve to the threshold level, can regeneratively trigger an action potential. In fact, action potentials are propagated down a nerve or across a muscle in just this fashion. The action potential at one section of a membrane renders the membrane highly conductive (the g in Figure 7) and also imposes a reversed polarity, both of which would tend to bleed the charge off the C_M of adjacent non-activated membrane. Once this C_M is bled down to the threshold level the adjacent membrane is regeneratively activated. The activation progresses onto the next adjacent piece of membrane by the same mechanism, and on until there is no more cell membrane to activate.

It is remarkable that in 1859 Pflüger had a foreshadowing of this in his "liberation hypothesis". He stated that nervous transmission was "not a simple advancing undulation in which the sum of the living forces is not increased" but a situation in which "new tension forces are set free by the living forces of the stimulus and become in turn living forces with each onward step" (11,p.22).

The description of events occurring in the neuronal (nerve cell) membrane has been discussed in considerable detail, because these studies form the basis of current attempts to understand the excitable behavior of muscle membrane. Unfortunately muscle cells with diameters as large as that of the squid axon are not available for the direct

experimentation such as that done by Hodgkin, Huxley and Katz. However enough direct evidence has been obtained from studies of the potential changes in muscle to ensure confidence in the essential similarity of membrane events in muscle and nerve. Figure 10 (53,p.343) illustrates an action potential of a skeletal muscle cell which is essentially similar to the neuronal action potential illustrated in Figure 6. This is not to say that all action potentials are the same. In fact, cardiac action potentials do show some significant differences in time course from those of skeletal muscle and nerve. These differences are still not completely understood, but it is assumed that the mechanisms underlying all action potentials are essentially similar. The present evidence in support of the applicability of the sodium hypothesis to cardiac cells is less convincing than that evidence which supports the theory for the squid giant axon. However, it seems extremely likely that an increase in P_{Na} plays a significant role in the action potential of most cardiac muscle fibers, but it also seems likely that other additional unknown factors are equally important (61,p.272).

The Electrocardiogram

The electrocardiogram (ECG) is a record of the potential variations on the surface of the body due to the electrical activity of the heart. The tissues of the body surrounding the heart are conductive (with a conductance similar to that of sea water). Therefore the action potentials of the individual cardiac cells are shunted by the surrounding conductive tissues, and this results in current-flow throughout this volume conductor. Obviously potential fields also exist throughout this volume

conductor, and more pertinently, they exist at the body's surface. The variations of these surface potentials (typically up to a few millivolts) can be detected with electrodes in contact with the skin. To make the source impedance of this potential as low as possible conductive jelly is customarily rubbed into the skin so that there is a relatively low resistance bridge between the metal surface electrode and the body fluids which constitute the volume conductor. Typically a surface electrode has a source impedance less than a few thousand ohms. Therefore the potential differences observed by instruments having high input impedances are truly the surface potentials resulting from the heart's electrical activity. By inference, these surface potentials are also indications of the mechanical activity of the heart. The clinical ECG is a record of such observations taken with the surface electrodes in various standardized positions, which will be discussed later.

The contraction of skeletal muscle is triggered by an action potential which dies out long before the resultant contraction subsides. With the occurrence of additional action potentials during the period of contraction, a skeletal muscle can be made to contract harder and continuously (physiological tetany). Cardiac muscle is unique in that this cannot happen since a cardiac action potential and its associated refractory period have a total duration that extends past the peak of contraction (87,p.92). Thus the heart cannot "lock-up" in a contracted state (i.e., cannot be tetanized). This is a built-in safety feature that ensures that each cardiac muscle contraction (beat) is followed by a period of rest during which inflow of blood may occur. Figure 11

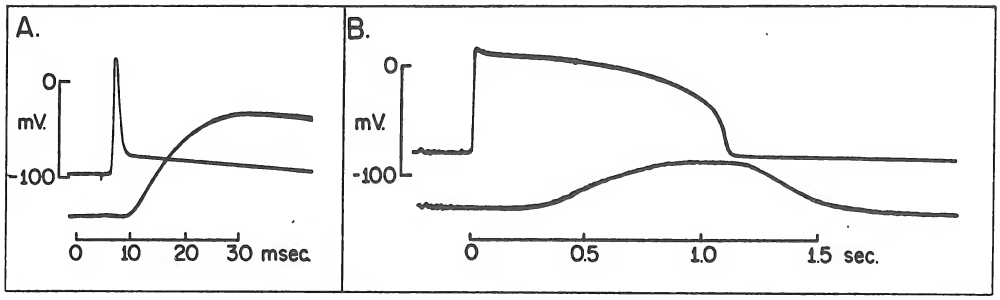


Figure 11. Comparison of action potential duration (above) and contraction timing (below). A.: skeletal muscle cell; B.: cardiac muscle cell.

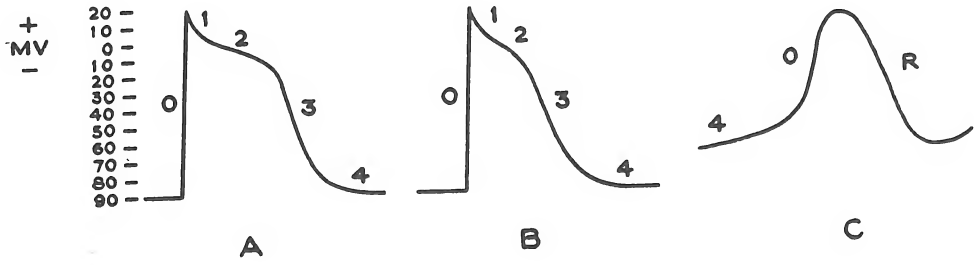


Figure 12. Diagrams of cardiac action potential curves. A: ventricular muscle cell; B: atrial muscle cell; C: S-A node. Numbers indicate phases. 0: rapid depolarization; 1: rapid repolarization; 2: plateau of repolarization; 3: slow terminal repolarization; 4: membrane resting potential; R: nodal repolarization is not divisible into phases.

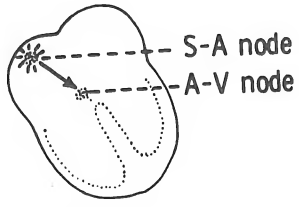


Figure 13. Activation of the atria. Conduction velocity throughout the atrial musculature is three times that of the ventricles. Activation terminates in the A-V node.

(131,p.118) permits the comparison of the action potential and contraction of skeletal muscle with that of cardiac muscle.

The rhythm of the heart beat is established by the not-so-well-publicized normal pacemaker. In the normal heart there is a specialized bit of cardiac muscle known as the sino-atrial or S-A node (for location refer to Figure 14). This piece of tissue has an intrinsic rhythmicity which causes it to behave somewhat like a relaxation oscillator; that is, it periodically initiates an action potential. Part C of Figure 12 (46,p.19.1) illustrates that the action potential of the S-A node has no base line. Therefore after the S-A node action potential subsides, its membrane potential, rather than resting, gradually approaches the firing threshold at 50 mV. Thus action potentials are spontaneously and periodically initiated by the S-A node.

The action potentials initiated by the S-A node sweep over the atria at the moderate speed of one m/sec (39,p.400). Thereby the atrial musculature contracts and forces additional blood into the ventricles. The electrical activity of the atria terminates at another specialized bit of tissue known as the atrio-ventricular or A-V node, which constitutes the sole conducting pathway between the atria and the ventricles. The spread of electrical excitation to the ventricles is momentarily arrested by the very slow conduction (0.1 m/sec) through this piece of tissue. This results in an A-V nodal delay of about 0.1 second which allows the atria time to complete the filling of the ventricles. Atrial activation is illustrated in Figure 13 (46,p.38).

The momentarily sustained squeeze-like contraction of the ventricles is due to two factors. First, all ventricular parts are almost

simultaneously activated (within 0.1 second) by the very fast (4 to 5 m/sec) conduction system that reaches all corners of the ventricular myocardium. This conduction system consists of modified muscular tissue specialized for rapid conduction. This system is illustrated in Figure 14 (46,p.38). Second the actual recruitment of ventricular fibers to the task is relatively smooth and gradual since the spread of activation throughout the volume of the ventricular muscle is rather slow (0.3 m/sec). The activation (depolarization) of the ventricles is illustrated in Figure 15 (46,p.39).

The depolarization of the atria and the ventricles results in the propagation of time varying potential fields throughout the volume conductor and to the surface of the body. Repolarization, of the return of the cardiac muscle fiber membranes to the resting potential, also results in time varying and detectable surface potentials. Figure 16 (14,p.28) is a highly schematized drawing illustrating the influence of depolarization and repolarization of a cardiac muscle cell upon the potential detected by the surface electrode P. Note that during the depolarization process the surface electrode sees dipoles predominantly of the form - +, and therefore the detected potential is positive. During repolarization the predominant dipole is + -, and the potential is therefore negative. Repolarization is a slower process than depolarization, and this results in a longer time course and lower amplitude for the repolarization waveform. The lower amplitude results from the fact that all parts of the muscle are more in phase with each other during repolarization, and thus the <u>differentially</u> detected surface

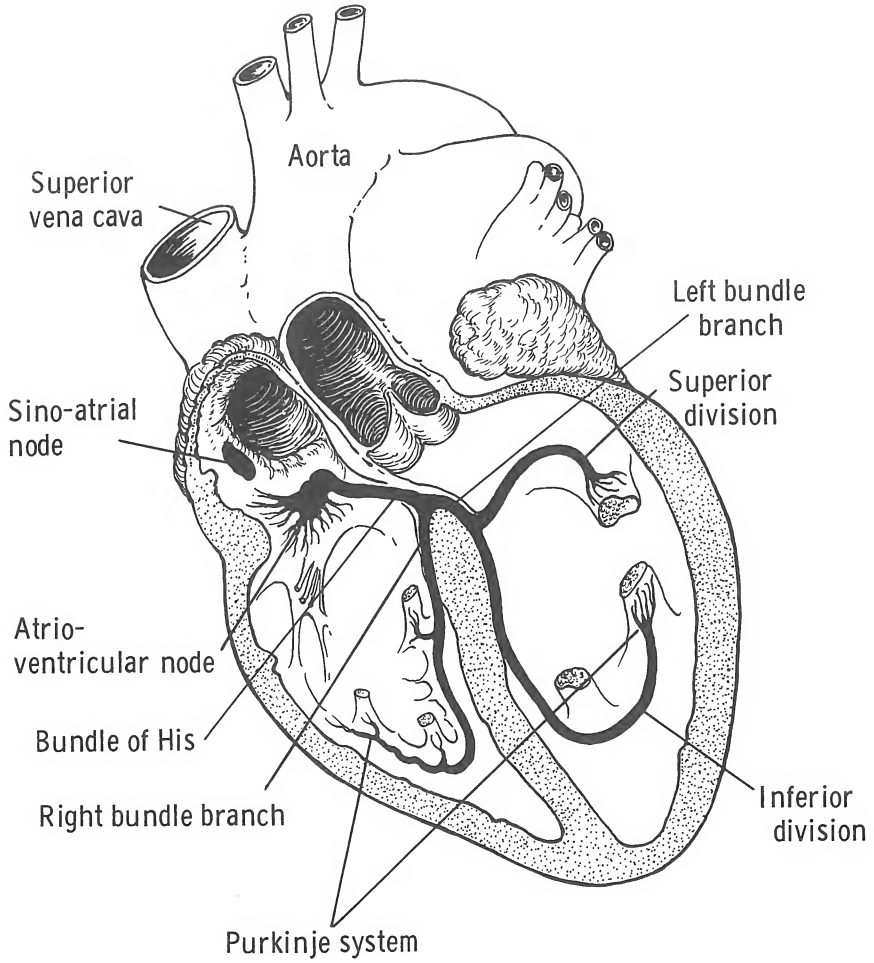
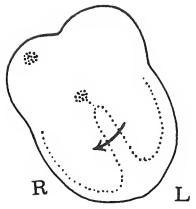
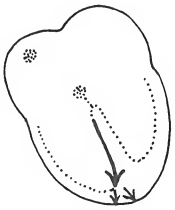


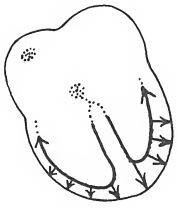
Figure 14. The conduction system of the heart consists of: the S-A node, atrial musculature, A-V node, bundle of His, left and right bundle branches, Purkinje system, and ventricular musculature.



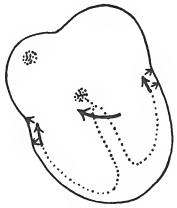
Septal activation from left to right.



Activation of anteroseptal region of the ventricular myocardium.



Activation of major portion of ventricular myocardium from endocardial to epicardial surfaces.



Late activation of posterobasal portion of the left ventricle, the pulmonary conus, and the uppermost portion of the I-V septum.

Figure 15. Ventricular activation.

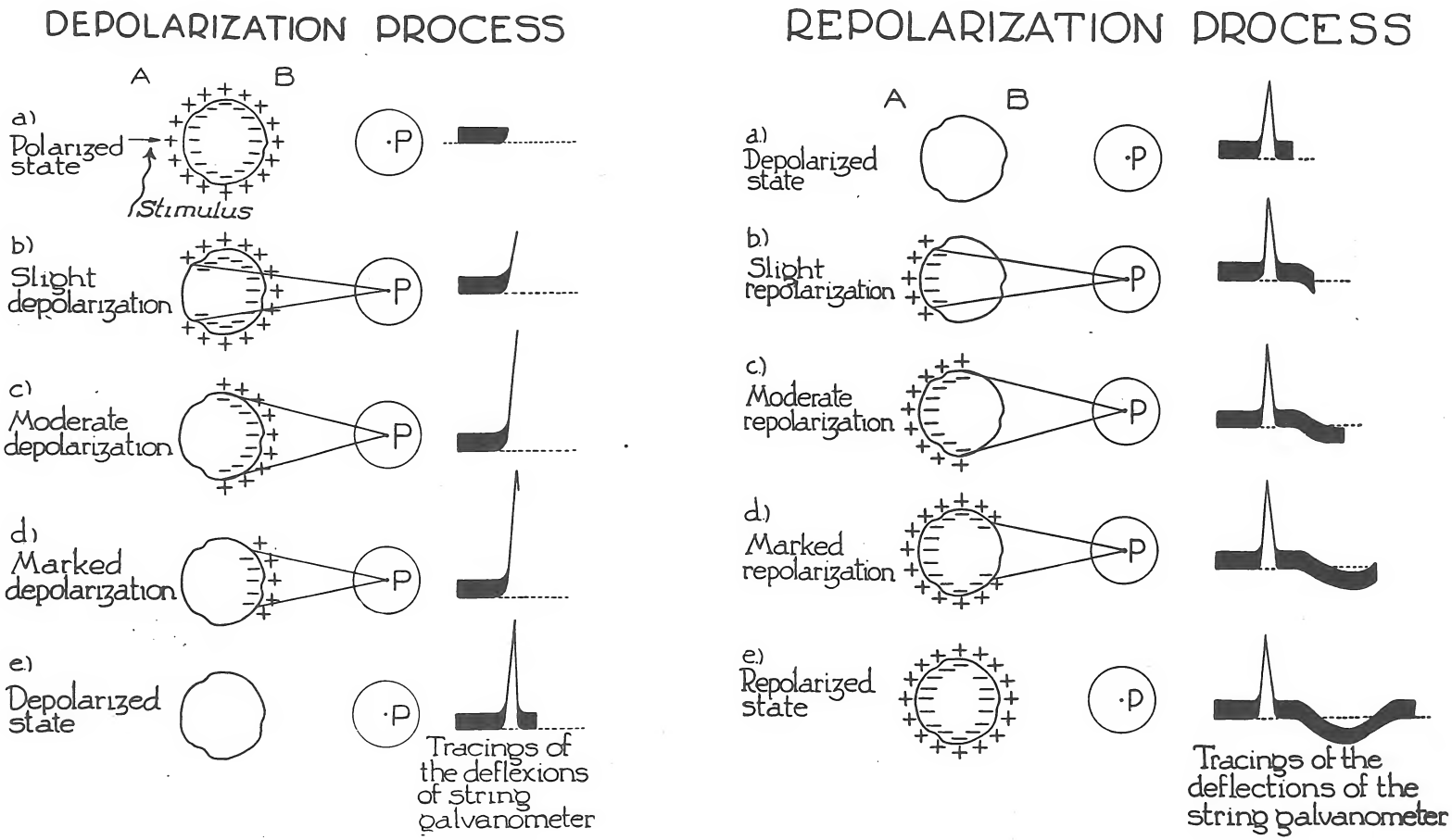


Figure 16. Schematized illustration of the influence of depolarization and repolarization of a cardiac muscle cell on the potential detected by an electrode P on the surface of the body.

potentials are of smaller amplitude. Since the total electromotive force variations over time are the same in both instances, the area included under each waveform is the same (14,p.29).

Actually a surface electrode detects the resultant potential due to the action potentials of all cardiac cells. The location of an electrode on the body surface fixes the view from which the electrode sees the electrically active heart. Different electrodes will not only see the heart from a different angle, but the heart will also be at a different distance and will intercept a different solid angle. Thus the various parts of electrodes will yield different cardiac waveforms. However the periodicity of the heart beat implies a like periodicity of the ECG waveforms, each cycle of which has distinctive landmarks. These sequential features were named by Einthoven, the father of electrocardiography, in a purely arbitrary fashion as P, Q, R, S, T, and U. Figure 17 (46,p.28) illustrates a typical ECG waveform with the distinctive features labelled. The P wave is the result of atrial depolarization. The QRS complex is made up of a Q wave, R wave and S wave, all of which result from ventricular depolarization. The R wave is the most distinctive feature of a typical ECG record, and it results from the <u>almost</u> simultaneous excitation of the ventricular myocardium. Repolarization of the ventricles results in the T wave. Atrial repolarization occurs at the same time as ventricular depolarization,and therefore the surface potential effects to be expected from atrial repolarization are buried under the ventricular action potential. The U wave, when it occurs is usually a positive deflection. The cause of this wave is not known, although it may be the result of after potentials

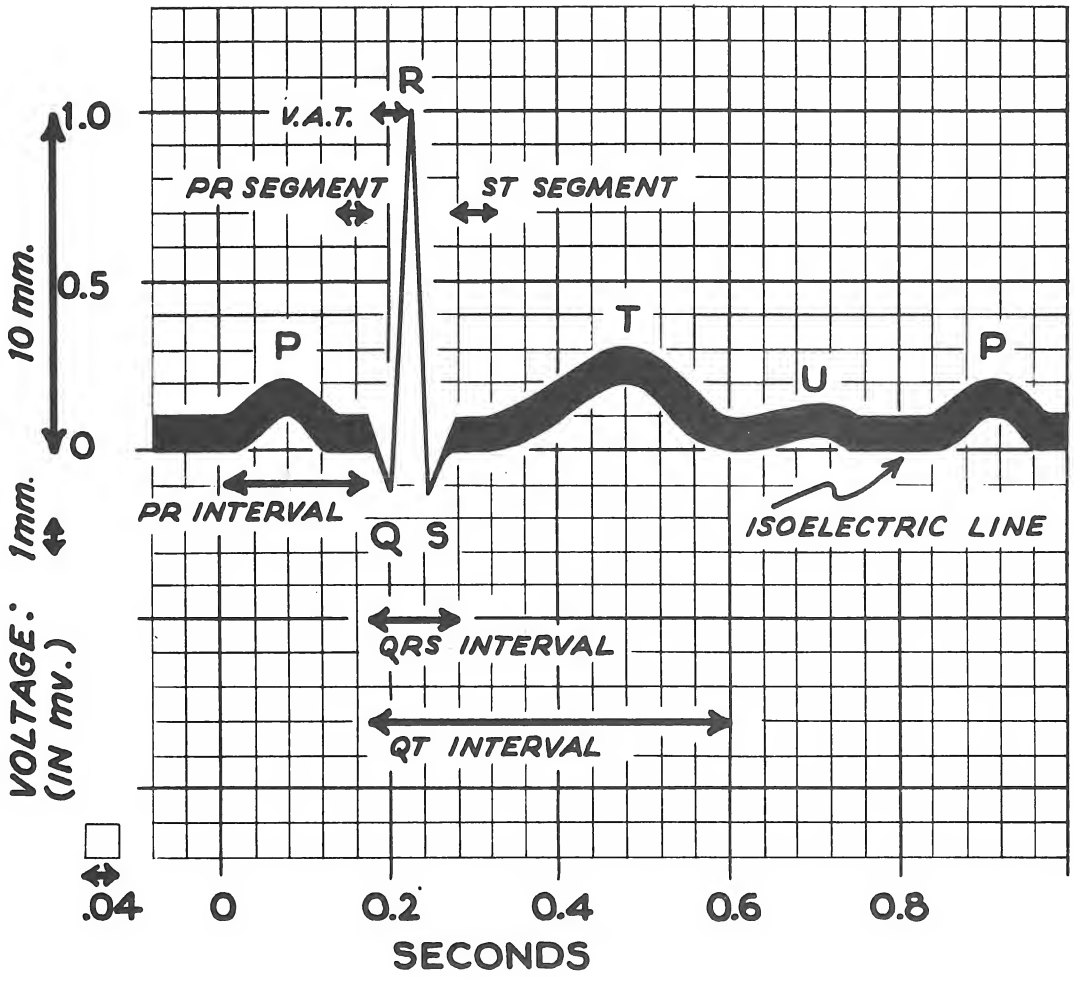


Figure 17. Diagram of electrocardiographic landmarks:

P wave: atrial depolarization,
QRS complex: ventricular depolarization,
T wave: ventricular repolarization,
U wave: possibly ventricular after potentials,
Isoelectric line: potential of the resting heart,
PR segment: end of P to Q (normally isoelectric),
V.A.T.: ventricular activation time,
ST segment: end of S to T (almost isoelectric),
PR interval: atrio-ventricular conduction time,
QRS interval: total ventricular depolarization time,
QT interval: duration of electrical systole.

(failure of the membrane to return to the normal resting potential) (46,p.25). The P-R interval is a measure of the atrio-ventricular conduction time, i.e., the time of atrial depolarization plus the conduction time through the A-V node. The QRS interval is the measurement of total ventricular depolarization time. The Q-T interval measures the duration of electrical systole.

The amplitudes and relationships of the above intervals and landmarks, plus observations as to timing, absence of waves, occurence of extra waves, etc. are the raw data that the clinical cardiologist uses in his interpretation of the ECG. The ECG is of particular value in the following clinical conditions (46,p.1): 1) atrial and ventricular hypertrophy (enlargement); 2) myocardial infarction (dead tissue and coagulated blood, e.g. a coronary thrombosis); 3) arrhythmias (non-periodicity of the heart beat); 4) pericarditis (inflammation of the sac supporting the heart); 5) systemic (bodily) diseases which affect the heart); 6) effect of cardiac drugs, especially digitalis and quinidine; and 7) disturbances in electrolyte metabolism, especially potassium abnormalities.

Lest the reader get the false impression that the ECG furnishes the complete answer to all questions about the heart (46,p.1):

> It must always be borne in mind that the electrocardiogram is a laboratory test only and is not a sine qua non of heart disease diagnosis. A patient with an organic heart disorder may have a normal electrocardiogram, and a perfectly normal individual may show non-specific electrocardiographic abnormalities. All too often a patient is relegated to the status of a cardiac invalid solely on the basis of some electrocardiographic abnormality. On the other hand, a patient may be given unwarranted assurance of the absence of heart disease solely on the basis of a normal electrocardiogram. The

> electrocardiogram must always be interpreted in conjunction with the clinical findings. In general, the person best qualified to interpret the electrocardiogram is the physician caring for the patient.

Development of Electrocardiography

Electrocardiography consists of the recording, by instruments, of surface potentials (or currents) resulting from the cardiac action potentials of the beating heart and the subsequent interpretation of these records. The first of these tasks can easily be done with suitable instrumentation and is the subject of this thesis. The second task requires a wealth of medical knowledge and experience and shall hardly be touched upon in the course of this paper. At one time the author had considered discussing the development of electrocardiography and the development of its instrumentation as two separate topics. However, they are so entwined historically, it is felt that a concurrent exposition gives a better perspective to these early examples of bio-medical engineering.

The 19th Century

Early Developments

It could be argued that electrocardiography was only possible after Oersted's discovery of electromagnetism in 1820 (95). This principle was, and still is, utilized in the construction of galvanometers for the detection of current and potential. The earliest forms were little more than a current carrying wire and a suspended magnetized

needle, which unfortunately was influenced by the earth's matnetic field. A useful form of the galvanometer was developed in 1825 when Nobili, Professor of Physics and Natural History at Florence, invented the astatic galvanometer which effectively cancelled the effect of the earth's magnetism by having two coils of wire wound in opposite directions (94). A similar type of galvanometer built in the United States by Locke in 1834 is illustrated in Figure 18 (81,p.105). Before long Nobili's usable galvanometer found its way into the laboratories of the Italian scientists studying animal electricity.

In 1843, Matteucci proved for the first time that there was an electromotive effect in cardiac muscle (64,p.1). Interestingly, he demonstrated this by stacking pieces of a pigeon's heart in series so that each cut surface of the cardiac muscle was in contact with the natural surface of the next piece. The end of this pigeon heart battery which presented the cut surface was electro-negative compared to the other undamaged end.

In 1856 Kollicker and Muller established the existence of action currents in the heart (72) by means of the physiological rheoscope (Gr. rheo = current + scope), which is a current sensitive frog muscle. When connected to the heart by a conductor the rapidly reacting skeletal muscle would contract before the heart would beat, thus establishing the precedence of the electrical events over the mechanical events in the cardiac cycle. They also made the pregnant observation that there were often two frog muscle contractions (electrical R and T waves?) for each cardiac systole.

It must be remembered that the action potential of a membrane

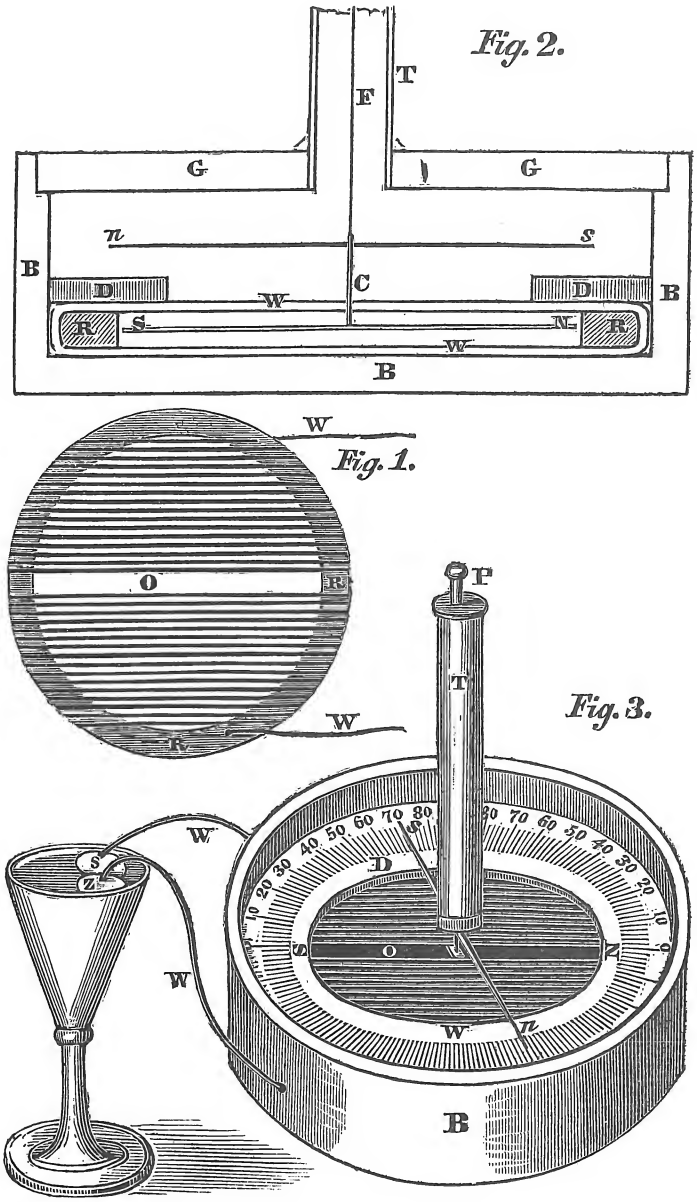


Figure 18. Locke's galvanometer. A ring of wood (R) is wound with wire (W) and is sealed in a box (B) with a compass card (D). The magnetized indicator (n-s) is suspended by a silk filament (F) within a brass tube (T). Beneath the indicator is the principle magnetic needle (N-S), parallel to the indicator, but of reversed magnetic polarity. It swings between the wires of the ring after having been inserted through the opening (O). A glass cover (G) supports the tube and eliminates agitation due to air currents. The battery consists of a glass of wine, a silver coin (S), and a piece of zinc (Z).

seldom exceeds 100 mV, and therefore the voltage variations available to an investigator at the surface of a muscle such as the heart would rarely exceed 10 mV. One would not expect that the galvanometers of the mid-19th century could detect the nuances of cardiac potentials. Even in that era engineers were fighting the gain-bandwidth problem. There were galvanometers that would detect millivolts, but unfortunately they had periods that were measured in seconds or tens of seconds. Risetimes were approximately one-third of the free periods of such instruments (no critical damping in those days), and, as we now know, action potentials are millisecond phenomena. Therefore it would seem that a technological impasse had been reached in the study of animal electricity. However no allowance has been made for the ingenuity of the frustrated investigator, when he suspects that a phenomenon is eluding him because of inadequate equipment.

In 1868, Bernstein, following up an idea of du Bois-Reymond invented the differential rheotome (7), an instrument which allowed him to extend the effective bandwidth of galvanometers and thus heralded the birth of sampling. By mechanically closing the galvanometer circuit for very brief intervals of time, the rheotome made it possible to sample the magnitude of the electrical activity at that very instant, independent of the very long periods of oscillation of sensitive galvanometers. A much improved version of the rheotome is illustrated in Figure 19 (17,p.434). By juxtaposition of the results of many measurements made at varying times after the initiation of the activity, he was able to fit together a representative picture of the action current.

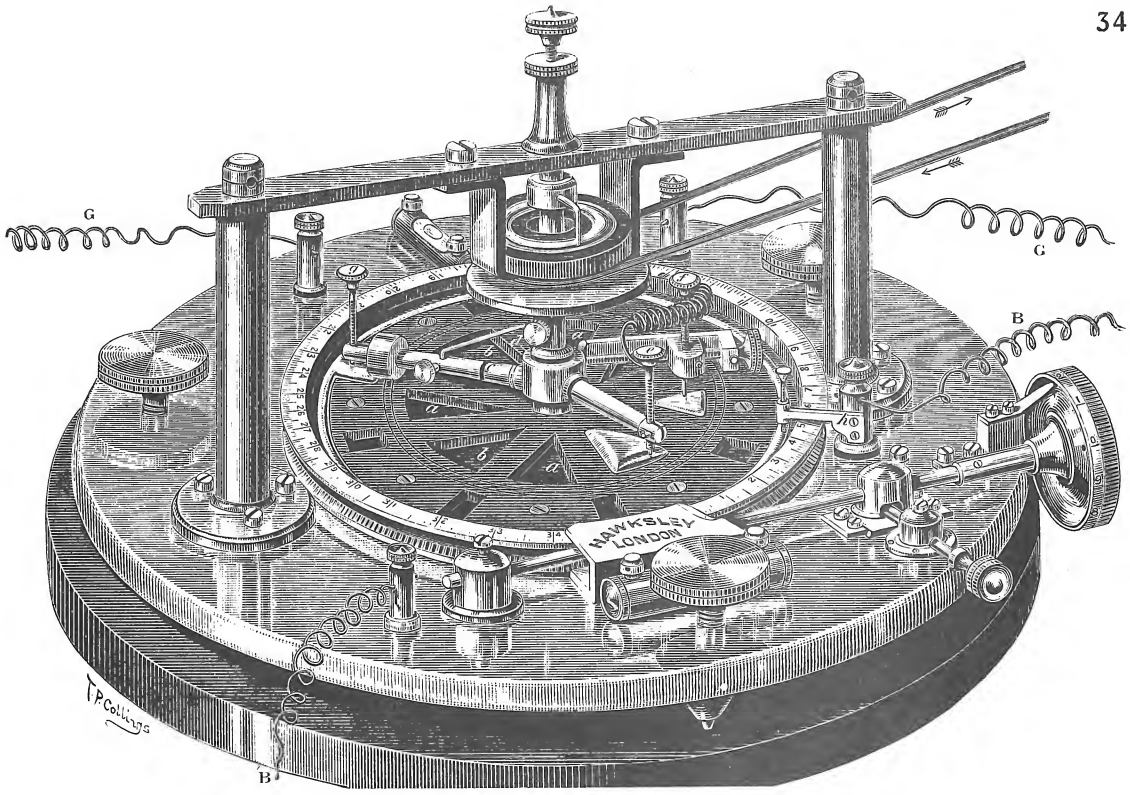


Figure 19. A highly developed differential rheotome. Circuits are opened and closed by contacts e,f, and g as they pass through pools of mercury located in sectors a,b,a,b,a, etc. The duration of closure is adjusted by the radius at which contact f is set and by the speed of rotation.

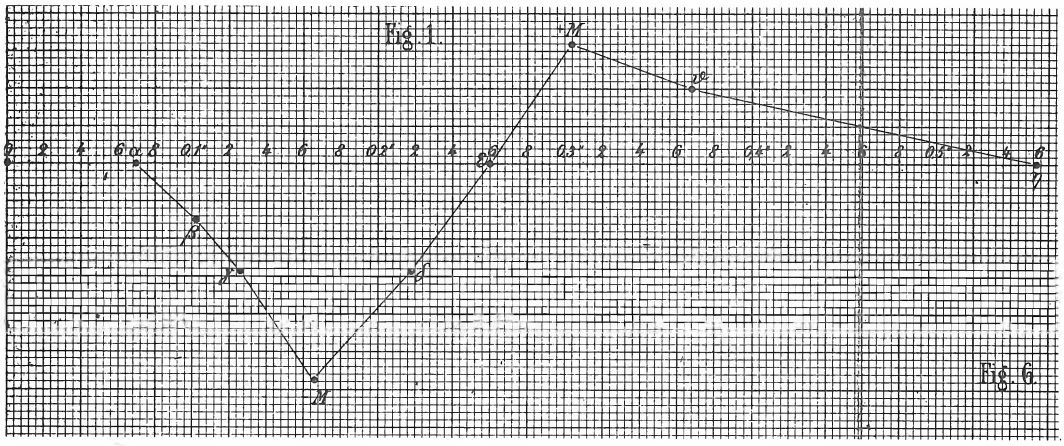


Figure 20. Cardiac action potential of a frog reconstructed by Th. W. Engelmann from differential rheotome measurements taken on February 26, 1874.

This technique was used extensively by the two investigators Marchand, who published first in 1877 (85), and Engelman, who started in 1874, but did not publish until 1878 (33). Both used the rheotome to quantitatively measure the frog's cardiac action current at given intervals of time after stimulation. These sampled measurements were equivalent to a series of points on a conventional ECG. Figure 20 (33, Table II) is one of Engelmann's reconstructions. From this data he concluded that there were two phases to the electrical variation (later named QRS and T): the first, brief and of relatively high potential, and the second, longer and of lower amplitude. He also finally gave in 1877 an explanation for the electro-negativity of damaged muscle, which he correctly interpreted as resulting from current of injury (64,p.1).

The Capillary Electrometer

Further developments were greatly aided in 1873 by Lippmann's invention of a more sensitive galvanometer, the capillary electrometer (79,80). As an operating principle this interesting instrument utilizes the electricly induced alterations of surface tension. A mercury-sulfuric acid interface has a surface tension which is sensitive to the migration of ions in such a way that current flowing in one direction increases the surface tension, while the reverse current decreases it. Therefore any difference between potentials applied to mercury and sulfuric acid will influence the surface tension at their interface. A capillary electrometer is illustrated in Figure 21 (92,p.19). Electrodes are attached via wires to the mercury in the capillary tube and sulfuric acid in the test tube. Surface tension acting as capillary

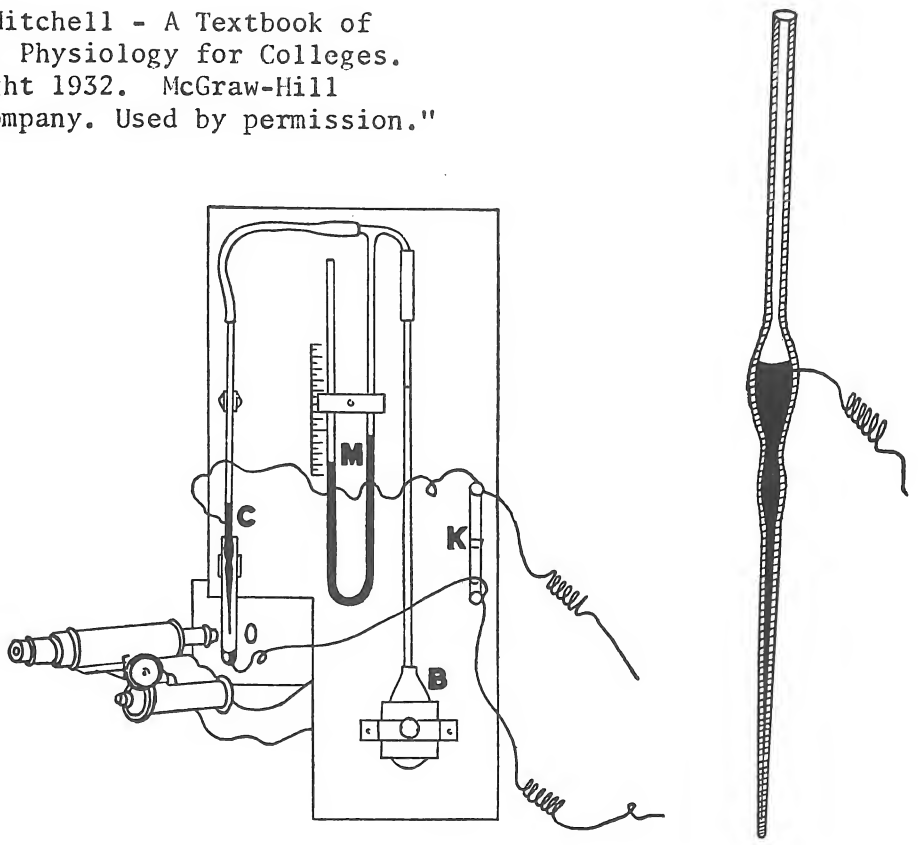


Figure 21. Lippmann's capillary electrometer. C, capillary tube containing mercury and having its lower end immersed in dilute sulfuric acid. A detail of the capillary tube is shown on the right. See text for principle.

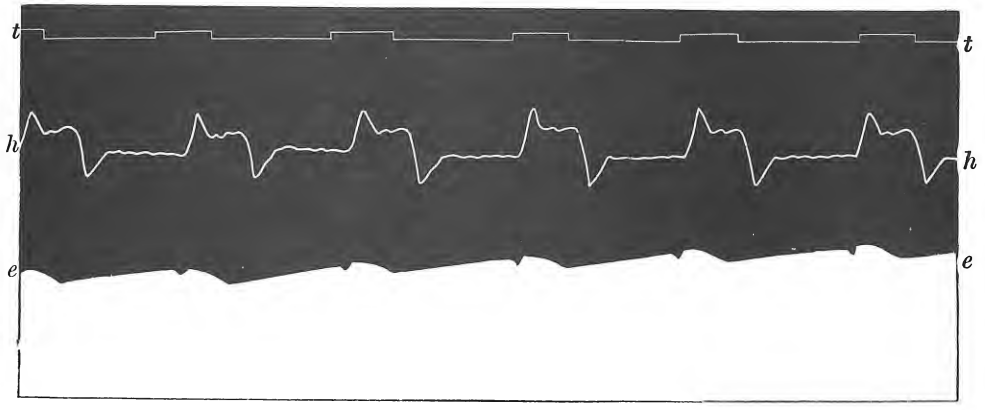


Figure 22. The first electrocardiogram, taken by Waller in 1887. t-t, time marks. h-h, cardiograph. e-e, electrometer.

attraction keeps the mercury from draining into the test tube. A microscope is used to observe the meniscus of the mercury. The bulb (B) controls the pressure in the system so that the meniscus may be brought into the field of view. The manometer (M) measures the pressure required to do this after current has displaced the column of mercury up or down.

In 1876 Marey introduced a recording technique which has been used extensively since that time. He projected the meniscus of the column of mercury onto a slit and photographed it by moving an unexposed plate past the back of the slit at a uniform velocity, thereby introducing a time axis. With this arrangement he succeeded in tracing the electrical phenomena occuring in the hearts of the frog and porpoise [? tortoise ?] during systole (64,p.2).

During this period outstanding contributions were made by Burdon-Sanderson and Page. Working with an improved rheotome (Figure 19) and the capillary electrometer they studied in considerable detail, and with photography, the electrical events occurring in the ventricles of the frog and tortoise, including such things as the effect of temperature on the timing and duration of electrical waves (16,17,18).

The First Electrocardiographer

Augustus Waller, an English physician, followed the lead of Burdon-Sanderson, and in 1887 he succeeded in taking the first ECG of man (118). Although not generally credited as such, he thus became the first electrocardiographer. By applying electrodes (zinc covered by chamois leather and moistened in brine) to the front and back of the

chest and using Lippmann's capillary electrometer and the photographic technique of Marey, he took the ECG which is reproduced as Figure 22 (118,p.229). The letters adjoining the figure have the following significance: t-t, one second timemarks; h-h, cardiographic level registering the motion of the heart's apex through the chest wall; e-e, the capillary meniscus. The shadow of the mercury is the white portion of the developed photographic plate and appears inverted when projected on the slit or when viewed directly through a microscope.

An enlargement of one of Waller's early records is shown in Figure 23 (134,p.190). The deflection of the electrometer meniscus clearly precedes the mechanical activity recorded by the cardiograph, thereby confirming earlier observations. It is clear that there are two electrical events associated with each systole. With measurements from this Waller deduced that the speed of activation of the ventricles was approximately five meters per second, a figure presently accepted. His investigations into the ECG of man resulted in the sophisticated diagram which is reproduced as Figure 24 (134,p.186). This is remarkably similar to numerous surface potential maps that have been produced during the last sixty years. Furthermore the electrical activity of the heart is implicitly represented as a spatially oriented dipole, a concept of which we will see more. Curiously, he could also be credited with the first usage of humans for bio-electric power. Figure 25 (134, p.192) illustrates the rather bizarre utilization of two human batteries as tandem pulse generators which, when synchronized, produced a double amplitude ECG to be recorded by the capillary electrometer.

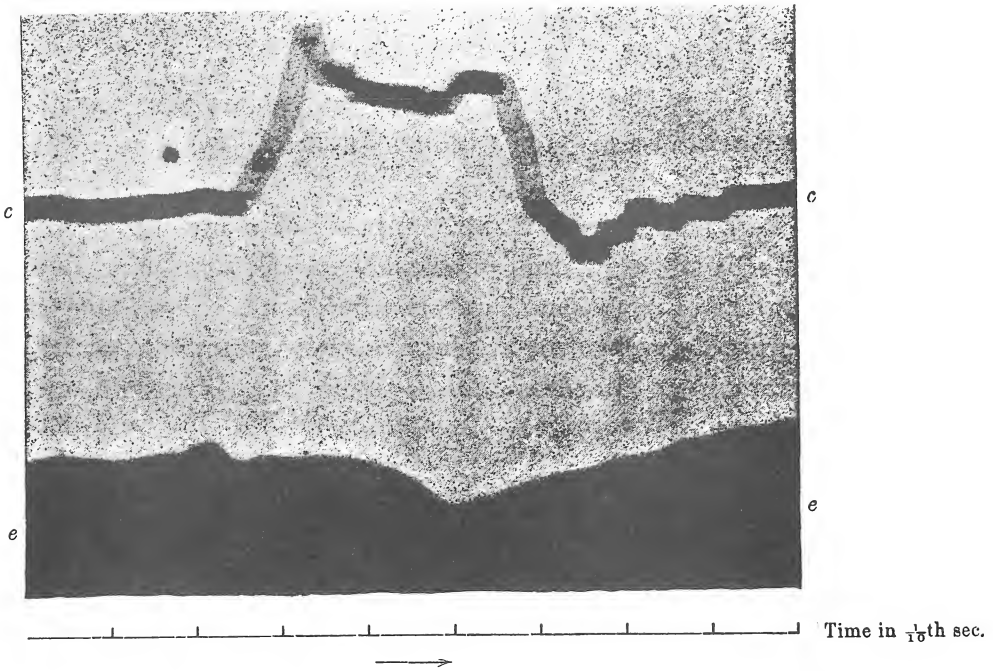


Figure 23. An enlarged ECG. c-c, cardiograph. e-e, electrometer. Note that electrical deflections precede mechanical.

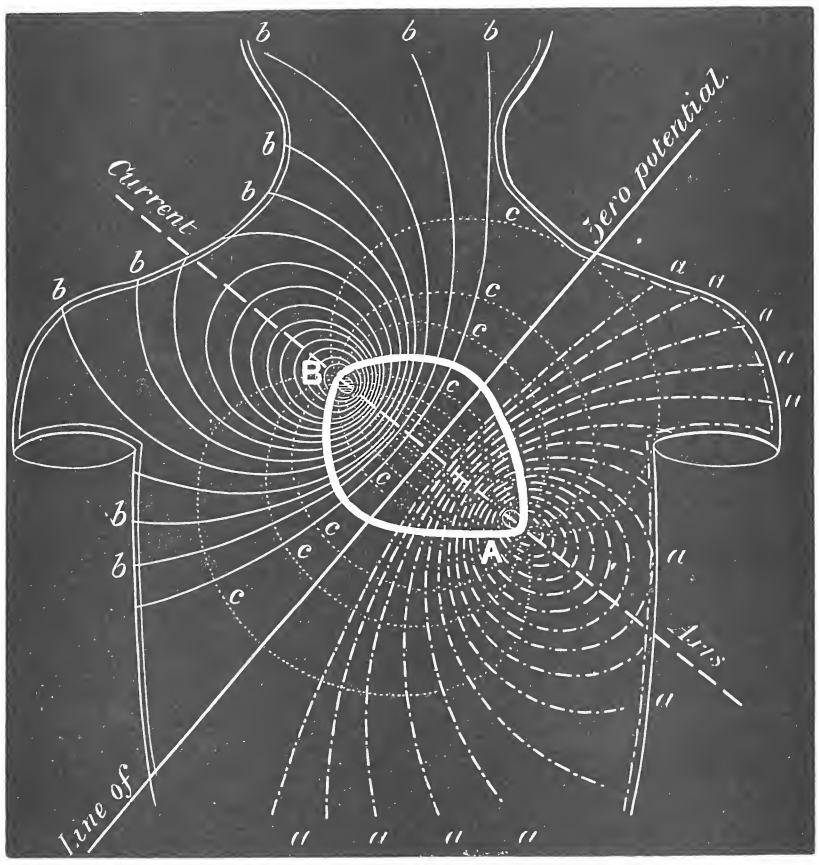


Figure 24. Waller's cardiac field diagram. The heart dipole BA causes equipotential lines a and b, and current diffusion lines c.

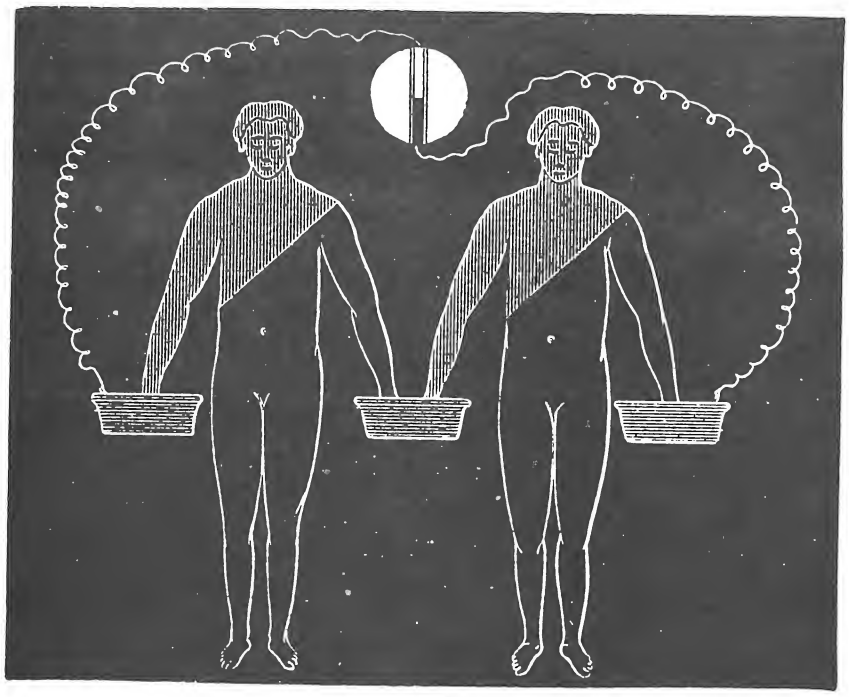


Figure 25. First utilization of men as a bio-electric power source.

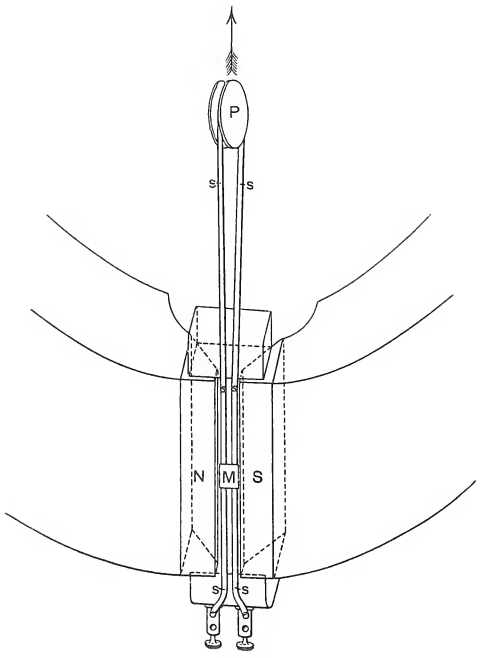


Figure 26. The Duddell oscillograph. Current flowing up and down the wire strips S causes the mirror M to twist in the magnetic field. An enclosing oil bath critically damps the system.

Galvanometers

Before leaving the 19th century it might be well to summarize the state of the galvanometric art. In 1882 J. A. d'Arsonval invented the well known type of galvanometer that has become the standard of the panel meter industry (40). Unfortunately its rather massive movement limits its frequency response to a few cycles per second. In 1897 Duddell (24) reviewed the state of oscillographs, i.e., those instruments capable of recording rapid (above a few cycles per second) variations of current or potential difference. He listed 27 different instruments roughly divided between sampling methods and continuous methods. Sampling instruments were divided into nine point methods, where the curve was plotted by hand, and four self registering methods, where the curve plotting was automated. The continuous methods were divided up according to what part of the galvanometer moved. Listed were four diaphragm, three soft iron vane, four wire coil, two plane polarized light, Braun's cathode ray tube, one stream of current carrying mercury, and three miscellaneous instruments: the capillary electrometer, a chemical method, and the induction oscillograph which attempted to compensate for the effects of mass and damping by means of induced currents from auxiliary circuits. Duddell then described his galvanometer which was designed to meet the following four criteria: 1) a movement of low mass, 2) critical damping, 3) negligible self induction, and 4) sufficient sensitivity. The resulting light galvanometer is diagrammed in Figure 26 (24,p.638). Its free period of vibration was measured at about 1/3000th of a second. At a screen distance of one meter its experimentally measured sensitivity was 1.13 cm per 0.1

ampere, which according to Duddell "fulfills the [sensitivity] condition very well." Therefore, it would seem that galvanometer of that day were markedly advanced as concerns frequency response, but not without the concomitant loss of sensitivity. Thus, at the turn of the century there were no high frequench oscillographs with the ability to detect the microampere currents resulting from the millivolt surface potentials of cardiac activity. The full potential of the Duddell oscillograph was not realized until the mid 1920's when the introduction of vacuum tube amplifiers provided the driving power necessary to extend the sensitivity.

One last significant galvanometer of the 19th century was a string galvanometer constructed in 1897 by Ader (64,p.2) for use in transatlantic telegraphy. An extremely thin copper or aluminum wire was placed in a magnetic field, and as current was passed through it, its deviations were photographed.

The Modern Era

Einthoven

There is no question that the modern era of electrocardiography began in 1903 with the successful refinement of the string galvanometer by Willem Einthoven, a Dutch physiologist (28). His much improved instrument combined both high sensitivity and an extended frequency response. Thus it was possible for the first time to record with acceptable accuracy the very small potentials which produce electrocardiograms, and significantly, in a routine manner. Figure 27 (14,p.2) is a

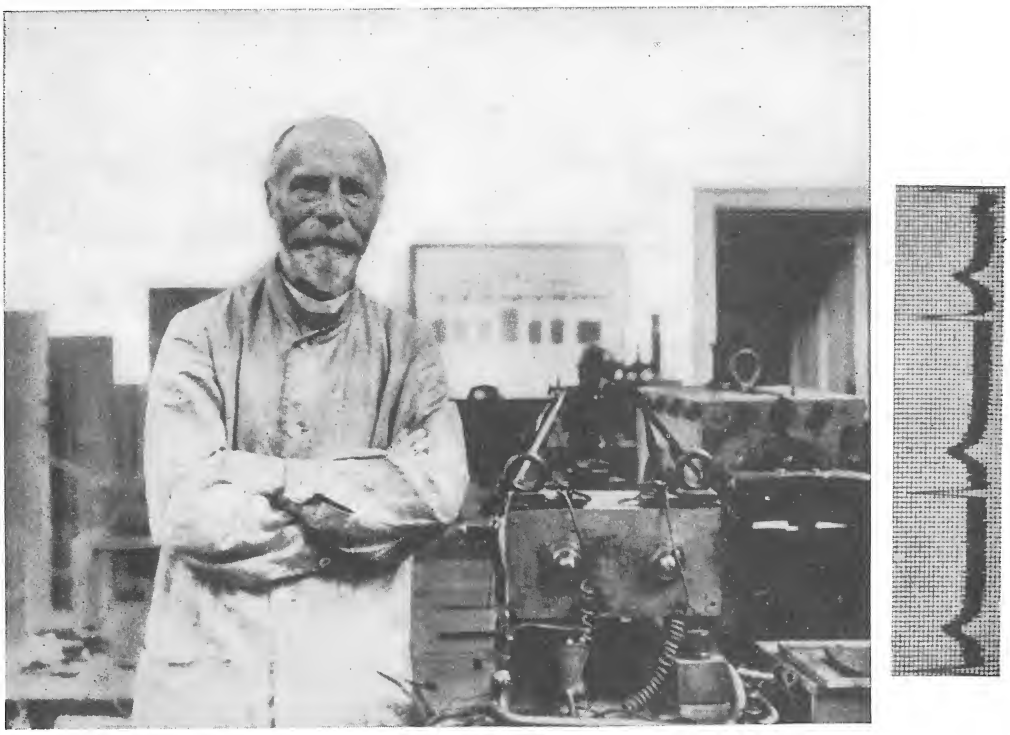


Figure 27. Willem Einthoven with his original string galvanometer in his laboratory, and a record made with this galvanometer.

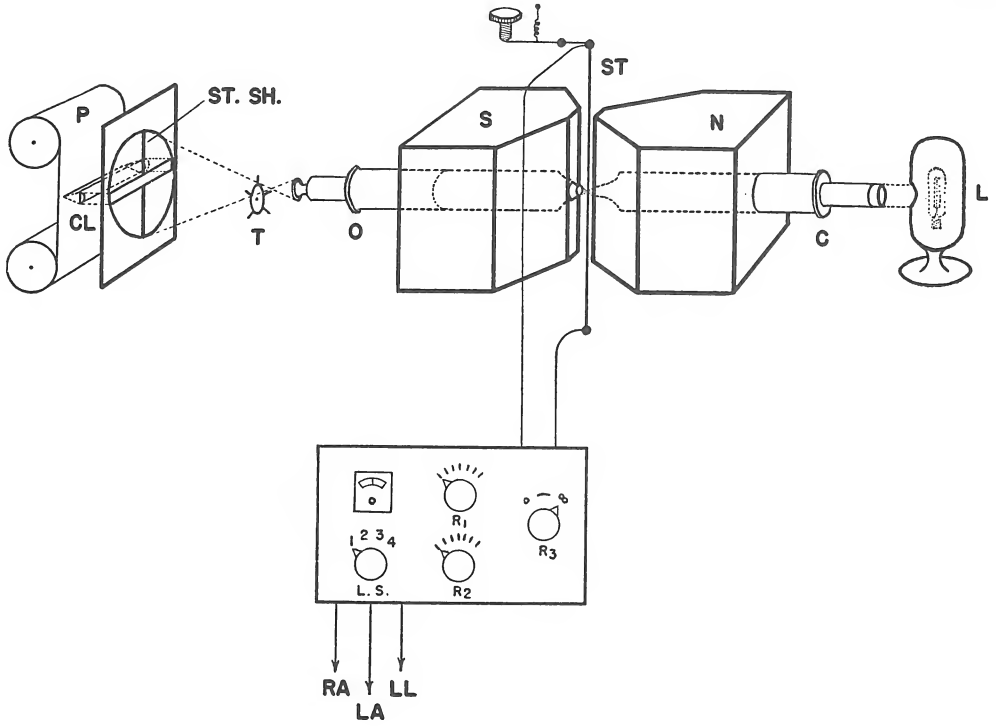


Figure 28. Diagram of the string galvanometer utilized as an electrocardiograph. Light from the lamp L passes through the magnifying optical system C-O, through a slit, and is then condensed into a fine line by the cylindrical lens CL onto the moving photographic paper P. The record is the shadow cast by the string ST deflections in the magnetic field N-S, the 40 ms timing wheel T, and the amplitude rulings of lens CL.

photograph of Dr. Einthoven in his lab along with an early ECG record. The principle components of an early electrocardiograph utilizing the string galvanometer are diagramed in Figure 28 (70,p.21).

The reader may well wonder how it is that a galvanometer, a low impedance current measuring device is used to measure accurately the very low surface potentials of the body. The galvanometer does load the body "circuit," and this does result in lower surface potentials. However the means of standardizing the string galvanometer deflections with the patient attached is done in such a manner that the recorded deflections are equivalent to the potentials that would exist on the patient, if the galvanometer were not attached (126). This technique may be illustrated by reference to Figure 28. The variable resistor R_3 shunts the galvanometer and is used to decrease sensitivity, while the variable resistor R_1 introduces compensatory current to center the string (which has been offset by the skin currents of the patient and/or the possible polarization of the electrodes). Then by moving R_3 to infinity, the galvanometer is brought to full sensitivity. The variable resistor R_2 in conjunction with the panel meter introduced into the circuit a standardizing current equivalent to one millivolt of surface potential. The galvanometer deflection sensitivity is then calibrated by adjusting the tension of the quartz fiber with the thumbscrew driven lever attached to the string ST.

By 1903 Einthoven had been studying the electrocardiogram for some years, for in 1895 he introduced the P, Q, R, S, T designations which are still in use today (26). At that time it was common practice to compensate mathematically for the inertia of the mercury column in the

capillary electrometer by reconstructing an inferred potential curve that would have been directly registered ("direct registrirten"), if measuring instfuments of that day had been able to respond fast enough. Figure 29 (26,p.105) illustrates the result of such a conversion, by calculation, of an electrocardiogram taken with a capillary electrometer into an inferred ECG. In essence, the "directly registered" ECG approximates the derivatives of the capillary electrometer curve. It was the inadequacy of the capillary electrometer response, necessitating these laborious calculations, which motivated Einthoven to construct the string galvanometer.

To attain both sensitivity and response Einthoven made two very significant advances in galvanometer design. To achieve frequency response he made the moving indicator of his galvanometer almost massless about one microgram (121,p.369)) by using an extremely thin (several microns (29,p.316)) quartz fiber which had been made conductive by a very thin deposition of silver (29,p.301). Typically this resulted in "strings" with resistances between two and ten thousand ohms. To extend sensitivity the current carrying silvered quartz fiber was first placed in the minute gap of a very powerful electromagnet: 20,000 gauss and sufficient flux to lift over 1100 pounds of iron (29,p.293). Next, the lateral deflections of the string were projected with a one thousand fold magnification by a coaxially located microscope in a boring through the middle of the electromagnet pole pieces.

The performances of such instruments are truly amazing. In fact, there has been no significant improvement in performance since the very first instrument. With the original instrument Einthoven was able to

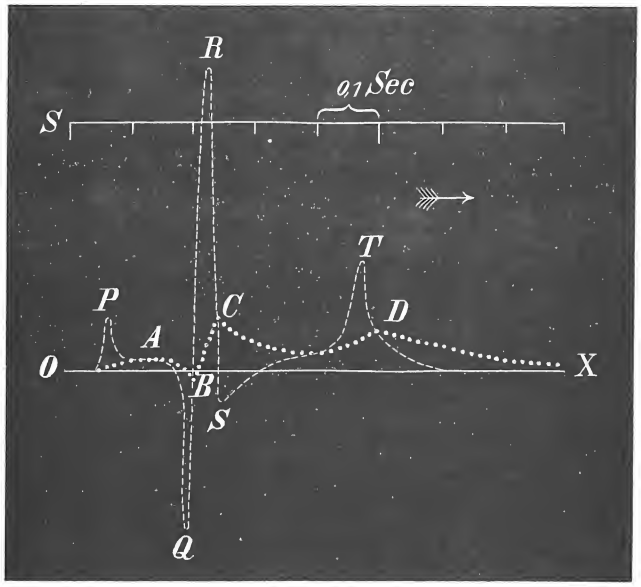


Figure 29. An 1895 reconstruction of the ECG (PQRST) from a capillary electrometer record (ABCD). Note that PQRST peaks are in phase with ABCD slopes. Also, terminology originated here.

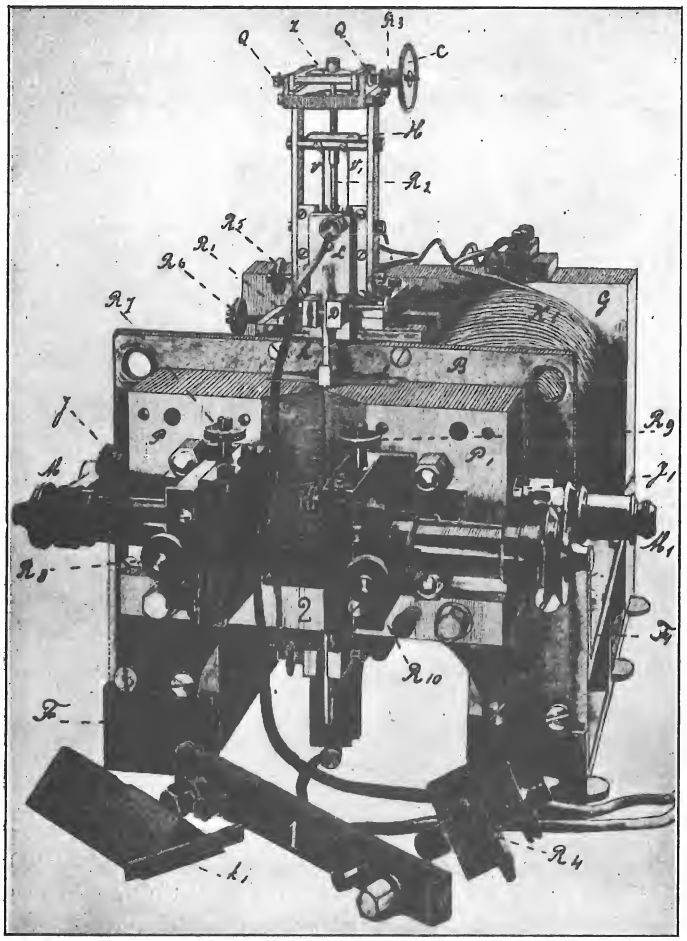


Figure 30. Einthoven's original string galvanometer, with keeper k_1 removed (bottom left) to allow insertion of the quartz string.

attain sensitivities of 5 x 10^{-11} ampere/cm (29,p.316), well above the 10^{-7} ampere/cm sensitivity which is standard for the taking of the ECG. Typically the free period of string vibration is between 0.1 and 1.0 millisecond (121,p.355-7), which is comparable to that of the Duddell oscillograph. However specialized forms of the string galvanometer when operated in a vacuum have attained natural frequencies as high as 300,000 cps (31). Einthoven's original string galvanometer is illustrated in Figure 30 (29,p.291). A commercial model suitable for ECG recording is illustrated in Figure 31 (29,p.320).

After having an instrument that would routinely and accurately record the human ECG, Einthoven did fundamental research into the technique of the recording, especially as regards lead systems: the number, location, and interconnection of patient electrodes. He devised a system of three electrodes: RA (right arm), LA (left arm), and LL (left leg) that is still used clinically today. ECG recordings were made between pairs of electrodes (three possible pairs), and the pairs were arbitrarily designated Lead I, Lead II, and Lead III. Figure 32, parts A and D illustrate the polarity convention (12,p.274). Einthoven realized that "there must be a connexion between the curves obtained by the three different leads from the same person. If two are known, the third may be calculated from them. The difference between the electrical tensions of Leads I and II must be equal to the electrical tension of Lead III. This may be formulated: Lead II - Lead I = Lead III" (30,p.854), and is known as Einthoven's Law. Today it is quite obvious that the ECG records are nothing more than records of the differential potential existing between the various patient electrodes, but this was

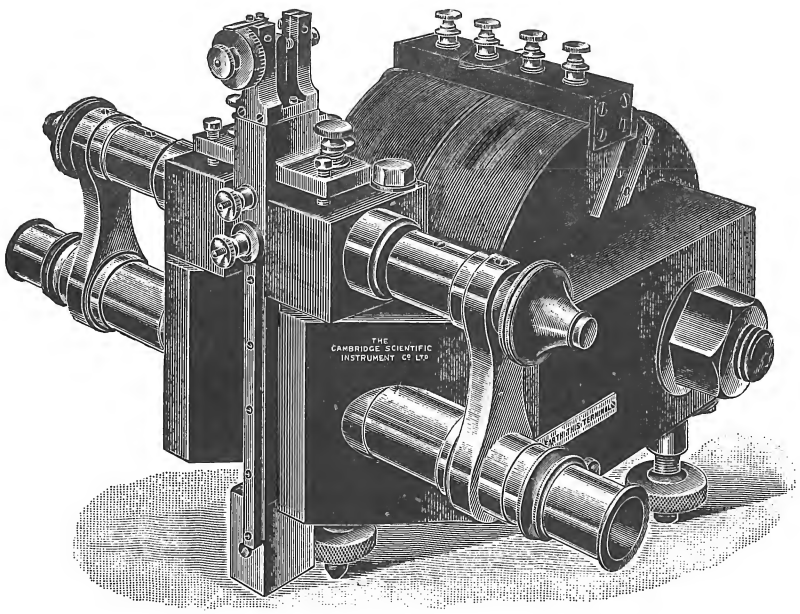


Figure 31. A commercial string galvanometer suitable for taking ECG's.

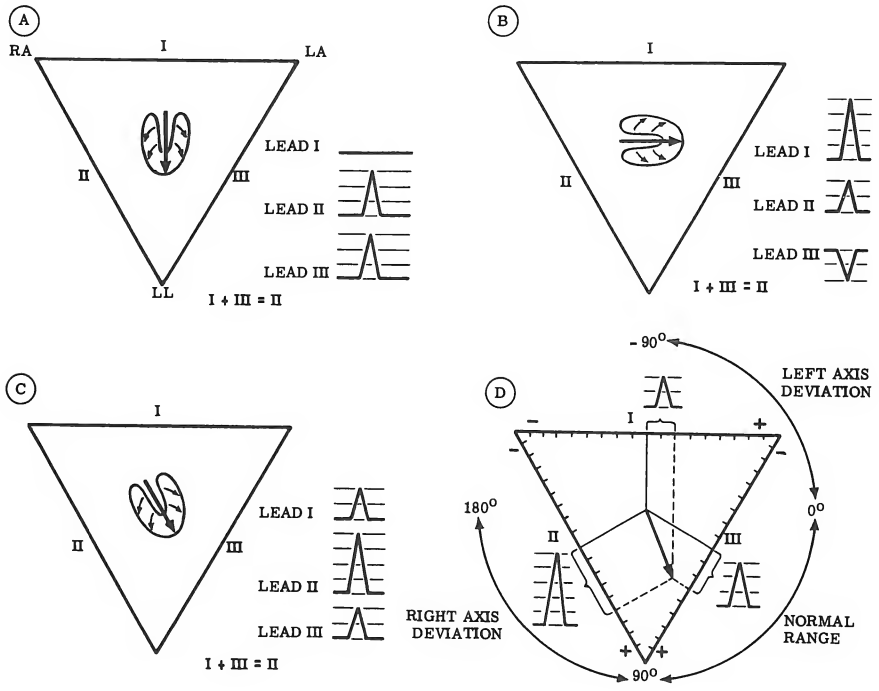


Figure 32. The Einthoven triangle hypothesis. Electrode locations are indicated in part A, while polarity conventions are shown in part D. In parts A, B, and C the ventricular myocardium is indicated by the horseshoe-like figure, ventricular activation by the small arrows, and the resultant manifest potential by the large arrow. Note the influence of ventricular position on the appearance of leads I, II, and III. Most of the population has an intermediate heart position, like C, with all leads +.

not appreciated theoretically until 1931 (126) and was not widely known about in clinical electrocardiography until about 1940 (47,p.13). Consideration of these differential potentials suggests an interesting explanation of Einthoven's law in terms of the potentials V_{RA}, and V_{LL} existing at the patient electrodes:

Lead I	+	Lead III	=	Lead II
$(V_{LA} - V_{RA})$	+	$(V_{LL} - V_{LA})$	=	$(V_{LL} - V_{RA})$
$-V_{RA}$	+	V_{LL}	=	$V_{LL} - V_{RA}$

Variations in waveforms to be found among the three leads had their first theoretical interpretation in Einthoven's triangle hypothesis (30, 32). First the human body is modeled as a flat homogenous plate (the frontal plane) in the form of an equilateral triangle. The electrodes RA, LA, and LL are at each corner of the triangle, while the heart lies at the center equidistant from the three electrodes. The instantaneous potential differences of the heart are assumed to have a net resultant effect that can be represented by an electric dipole (or manifest potential) vectorially oriented at the center of and in the plane of the triangular plate. Then the projection of this manifest potential vector on each side of the triangle is the amplitude of the instantaneous potential recorded in the lead of that side. This is very nicely shown in part D of Figure 32. Conversely working from any two of the recorded leads I, II and III it is possible to determine for any instant the direction and amplitude of the manifest potential. Obviously this hypothesis assumes that there is no loading by the electrocardiograph, and that the mass of the body is homogeneous so that the resistances to

each electrode are equally great. Furthermore it is assumed that an electrode on an arm or leg is equivalent to an electrode at the corner of the hypothetical triangle, and that since a single point LL represents both feet, it is assumed that either foot would give identical results. Obviously the above assumptions are only approximately met, yet they are sufficiently close to validate a workable hypothesis of considerable power.

This schema was used by Einthoven to investigate the variations of the P, R, and T waves by means of the manifest potentials existing at their summits (P_m, R_m, and T_m). He was able to point out how respiration, body position, heart rate, and pathological conditions could influence P_m, R_m, and T_m. Thus "by means of the schema one is in a position to separate from each other the real changes in the heart's action and the apparent ones, which are caused only by changes in the position of the heart" (32,p.303). For his many contributions, Willem Einthoven is justifiably called "the father of electrocardiography."

Clinical Electrocardiography

By 1908 a few clinicians had commercial instruments by which they could investigate the electrical activity of the heart, especially as to the effects of pathological conditions. Before long there was a publication explosion; an abridged bibliography of the heart published in 1929 had 137 entries under "Electrocardiography" (25,p.780-4)! Clinical electrocardiography developed rapidly in the 1910's and '20's under the leadership of the London school led by Sir Thomas Lewis. He contributed much to both theoretical research and clinical practice,

especially in the areas of arrhythmias and the process of ventricular activation (77,78). His papers are classics in the development of our understanding of electrophysiology and electrocardiography. His reasoning is lucid, his expression exact, and his contributions must be read by any serious worker in the field. The voluminous nature of the literature and the author's inexperience precludes any serious attempts to discuss comprehensively the clinical aspects of electrocardiography. Henceforth this aspect will be excluded except insofar as it impinges upon the concept or the instrumentation of the ECG.

However, one aspect of clinical electrocardiography that is pertinent to this paper is the electrical connections to the patient. One of the serious difficulties encountered by one first delving into this subject is the multiplicity of electrode configurations (all, however, specifically defined) and the multiplicity of the jargon associated with them (usually combinations of letters and numbers). Furthermore a person accustomed to dealing with electrical hardware is often "bugged" by the confusing aspect of the terminology which uses the word "lead" to mean and/or all things external to the recording apparatus; that is, the particular measuring concept, the electrode sites, the electrodes, the hook-up wires, any interconnections or resistive matrixing, or any external thing that influences the recording.

Precordial (L. - before the heart) leads are electrode arrangements whereby one electrode (the exploring, precordial, or chest electrode) is placed on the chest, while the other (indifferent) electrode is usually placed as far from the electrical influence of the heart as is possible. Bipolar leads are leads in which both electrodes have an influence upon

the record, e.g., leads I, II and III. Unipolar leads are leads in which only one electrode has influence upon the record, the other electrode completes the circuit for the electrocardiograph, but is located at a site where the potential variations are minimal.

The first electrode arrangement used in electrocardiography was a bipolar precordial arrangement, since in 1887 Waller had used electrodes on the front and rear of the chest. In 1909 Nicolai suggested the unipolar concept whereby an indifferent electrode was to be removed from the electrical sphere of influence of the heart. Unfortunately such an electrode site was not to be found until several decades later. Einthoven was very familiar with the fact that the potentials found anywhere upon a limb are essentially identical, and that there is therefore no indifferent site on a limb regardless of how far the location may be physically removed from the heart. As he was also familiar with the differential nature of the records made with his string galvanometer, he undoubtedly realized the bipolar nature of leads. These realizations may have influenced him to adopt the bipolar I, II, III lead schema which does have the attributes of being simple, symmetrical, consistent, convenient with patients, and not too difficult to interpret in terms of the triangle hypothesis. In any case little clinical attention was paid to precordial leads in the 1910's and '20's. However Lewis did publish several experimental studies dealing with "direct leads" placed in contact with the hearts of animals: these leads were, of course, bipolar.

Wilson and His Central Terminal

In 1930 Frank N. Wilson, one of the great electrocardiographers of all time, pointed out his difficulties in attempting to extend the triangle hypothesis of Einthoven into three dimensions via electrodes placed at the apices of a tetrahedron located within the human trunk (see Figure 47). The nub of his problem was in the placement of the electrodes. The triangle hypothesis gives consistent results because it is relatively insensitive to electrode placement. Since an entire limb is at essentially the same potential, it makes little difference whether the electrode is on the hand, wrist, forearm, or upper arm. However with the trunk tetrahedron variations of a few centimeters in electrode position have a profound effect on the record. As a result he could place no confidence in the manifest potential projections as initially applied to his tetrahedron. He concluded that the sensitivity to electrode placement was due to the proximity of the heart. "The potential variations of the electrode which is placed close to the heart will not only be very much greater than those of distant electrode, but they will represent the activity of various portions of the heart unequally" (122,p.612).

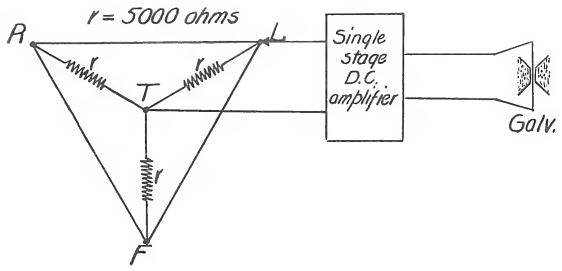
From this the concept of the unipolar exploring electrode grew. But for a short while there were the "semi-direct" leads (112,p.613):

> When one electrode is placed upon the precordium and the other at a point relatively distant from the heart, the precordial electrode is much nearer the anterior wall of the heart than the posterior; consequently the electrical activity of the anterior wall of the heart has a much greater effect upon the form of the curve than the electrical activity of the posterior wall, just as the subadjacent muscle in the case of direct leads. Leads in which one electrode is placed close to the heart are

> therefore semi-direct leads. It is not surprising that the curves obtained from such leads are in many respects similar to those obtained by placing one electrode upon the exposed heart. In both cases the position of the second electrode, so long as it is placed upon a point distant from the heart, has comparatively little effect upon the form of the curve recorded. When one electrode is placed upon the ventricular surface and the other upon a distant point, the arm or leg, for instance, deflections are obtained which have a value of 40 to 80 millivolts, a value approximately 20 times that of the tallest deflections that occur in the standard leads. Since the arm and leg do not show a difference of potential exceeding three or four millivolts at any time during the cardiac cycle, which extremity is used as the distant point is relatively immaterial. Consequently, when one electrode is placed upon the heart and the other upon one of the extremities the resulting curve is, for all practical purposes, a record of the variations in potential of the electrode placed upon the heart. The potential variations of a point on the precordium are very much smaller than the potential variations of a point upon the heart; they are still five to ten times as great as the potential variations which occur at points upon the extremities.

Even though the potential variations of the indifferent electrode only had a small effect upon the precordial records, they were nevertheless a nuisance. In 1931 Wilson and his associates devised a method whereby the potential variations of a limb could be calculated from the records of the I, II, III leads (127). By subtracting this variation out of a precordial lead record, they succeeded in producing a record of the potential variations detected by the precordial electrode alone.

Further progress at this point required a nudge from the radiotechnology that had blossomed in the 1920's. Vacuum tube amplifiers with high input impedances made it possible to detect the actual surface potentials. Up to this time surface potentials had been inferred from surface currents by means of the galvanometer calibration. This allowed Wilson and his associates to construct in 1932 a central terminal (128) that averaged the limb potentials V_R, V_L and V_F. Figure 33 (123,p.448)



"From Wilson: American Heart Journal 9:447, 1934, published by the C.V. Mosby Company."

Figure 33. Wilson's central terminal T being used with L as Lead V_L.

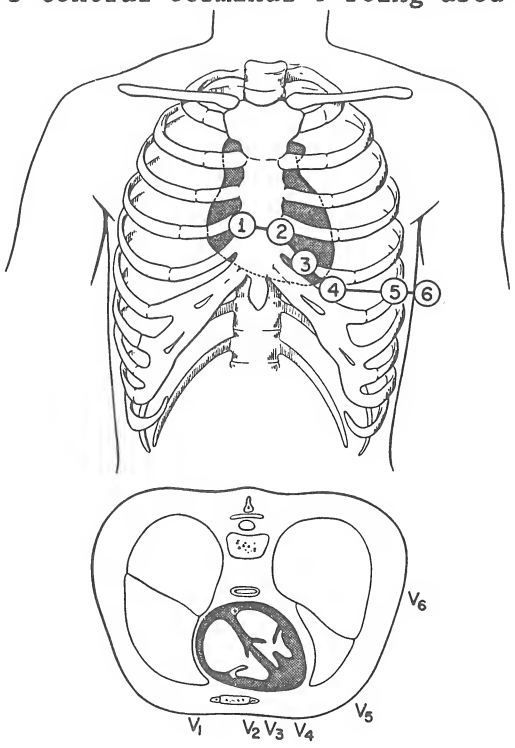
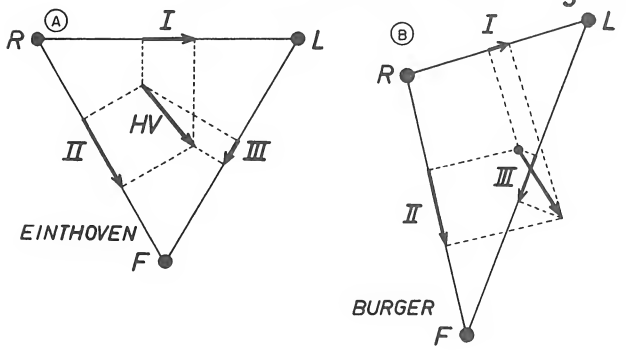


Figure 34. Position of unipolar precordial (chest) leads as routinely recorded in electrocardiography. V_1 and V_2 in fourth intercostal space at edge of sternum. V_4 in fifth intercostal space at midclavicular line. V_6 in fifth intercostal space at midaxillary line. V_3 between V_2 and V_4; V_5 between $V_{4,6}$.



"By permission of the American Heart Association, Inc."

Figure 35. Einthoven's triangle assuming a homogeneous torso, and Burger's triangle with the effects of the lungs and spine.

illustrates the connections of the central terminal T. The value of r = 5000 ohms is a compromise between the high impedance desired to minimize the effects of variable skin resistance and the low impedance necessary to minimize 60 cycle pick-up. In a previous publication (127) Wilson and his associates showed that if the assumptions of the triangle hypothesis (the heart in the plane of the limb leads, and the heart centered in the triangle) were true, then the limb potentials could be given by:

$$V_R = -(e_1 + e_2)/3 \qquad V_L = (e_1 - e_3)/3 \qquad V_F = (e_2 + e_3)/3$$

where e_1, e_2 and e_3 are the potentials of leads I, II and III respectively. It is easily seen that the sum of these potentials is zero.

$$V_{CT} = V_R + V_L + V_F = 0$$

Here at last was the site first spoken about Nicolai in 1909, a location removed from the sphere of the heart's electrical influence (93).

Other Workers

In 1932 Wolferth and Wood published the first clinical paper on the use of the precordial leads (129). Over the next few years they organized the use of these leads for the clinician, drawing heavily upon the experimental studies previously done by Lewis and Wilson. Unfortunately their precordial leads were usually recorded in a fashion which is now considered to be upside down. In 1938 a committee representing the American Heart Association and the Heart Association of Great Britain and Ireland standardized the precordial lead technique. They reversed the galvanometer connections of Wolferth and Wood so that when the precordial electrode is relatively positive an upward wave is written on the

record. They also attempted to standardize chest lead nomenclature (14,p.122).

The standard leads are the I, II, III leads and have been discussed previously. For other leads the following polarity conventions are in use. The indifferent electrode is connected to the negative input of the recording apparatus. The exploring electrode is connected to the positive input, and therefore when it is relatively positive there is an upward deflection of the recording. The unipolar precordial lead is taken with the exploring electrode placed over the apex of the heart regardless of its position (14,p.125), while the indifferent electrode is variously placed on the foot (F), right arm (R), left arm (L), back (B), or Wilson's central terminal (V). This precordial lead is often called Lead IV, and has led to the nomenclature IV_F, IV_R, IV_L, IV_B, IV_V depending upon the location of the indifferent electrode. The lead has also been designated as 4_F, 4_R, 4_L, 4_B or 4_V. This precordial lead is also sometimes called the chest lead with the nomenclature C_F, C_L, C_R, etc. The advent of Wilson's central terminal made it possible to obtain the unipolar limb leads, whereby the negative electrode is connected to the centrol terminal and the positive exploring electrode is connected to the right arm, left arm, or foot, yielding V_R, V_L, V_F. By moving the precordial electrode to different areas of the chest it is possible to record multiple precordial leads. Figure 34 (110,p.593) illustrates specific sites that have been conventionally agreed upon. These positions are called the CF, CR, CL, CB, or V leads and carry a numerical subscript that indicates the specific site for the exploring electrode. For example, the V_4 leads means that

the indifferent electrode is at the central terminal and that the exploring electrode is at position four, which is in the fifth intercostal space in the left midclavicular line, which in the normal subject is usually just to the left of the heart's apex.

In 1942 Goldberger introduced a new type of lead (42). He constructed a central terminal with just wire and without the equalizing effect of the 5000 ohm resistors. He concluded that (42,p.491):

> for purposes of clinical electrocardiography, it is not necessary to equalize the resistances of the circuit by the introduction of fixed resistances (the Wilson assembly); the three extremities may be joined to a central terminal with ordinary electric wire, and the Wilson assembly and the author's indifferent electrode may be used interchangeably in the recording of precordial leads.

He also pointed out that ordinary unipolar extremity leads (V_R, V_L, V_F) could be taken in the same manner, but that by removing from the central terminal the connection to the extremity that was being measured an augmented record (aV_R, aV_L, aV_F) with a 50% increase in amplitude could be obtained. Consideration of the differential nature of the recordings makes it clear that removing the lead from the measured extremity just converts the common mode signal of that extremity into a differential signal. Since the central terminal potential is approximately $(V_R + V_L + V_F)/3$ and the potential of the exploring electrode on the right arm would be V_R, the differential signal when recording lead V_R would be:

$$V_R - 1/3\ V_R - (V_L + V_F)/3 = 2/3\ V_R - (V_L + V_F)/3$$

However with the Goldberger method the lead would measure the potential

$$V_R - (V_L + V_F)/2$$

which is precisely 3/2 times the lead V_R.

In 1947 Goldberger summarized the bases and results of the unipolar

leads (43) and thereby started their widespread clinical use (64,p.3). Today the leads ordinarily taken in a routine electrocardiographic examination are I, II, III, aV_R, aV_L, aV_F, V_1, V_2, V_3, V_4, V_5, and V_6.

In 1947 Burger pointed out the well known, but ignored, fact that the human torso is neither homogeneous nor triangular and that this leads to a distortion of the electrical fields. Therefore manifest potentials deduced from Einthoven's triangle would be in error. Burger made allowances for the inhomogeities of the torso by the introduction of a distorted triangle. For the reader's comparison Einthoven's and Burger's triangles are illustrated in Figure 35 (106,p.338).

Instrumentation

Instrumentation has also made progress during the sixty years since Einthoven's first string galvanometer. Not long after his investigations into leads (1907-1908) there were several manufacturers of electrocardiographs in England and on the continent. All instruments consisted of Einthoven's basic apparatus: a string galvanometer, an optical system, and a moving film camera for recording. Figures 36 through 38 illustrate the progressive refinement of electrocardiographs through the 1910's, '20's, and '30's. Today's small metallic surface electrode is also the result of evolution as noted by the electrodes in Figure 39 (77,p.6), Figure 40 (120,p.93), and Figure 69.

The introduction of vacuum tube amplifiers in the 1920's made possible another type of electrocardiograph. By a 3,000 times amplification of the surface potentials, sufficient power was made available to drive the relatively massive movements of galvanometers such as

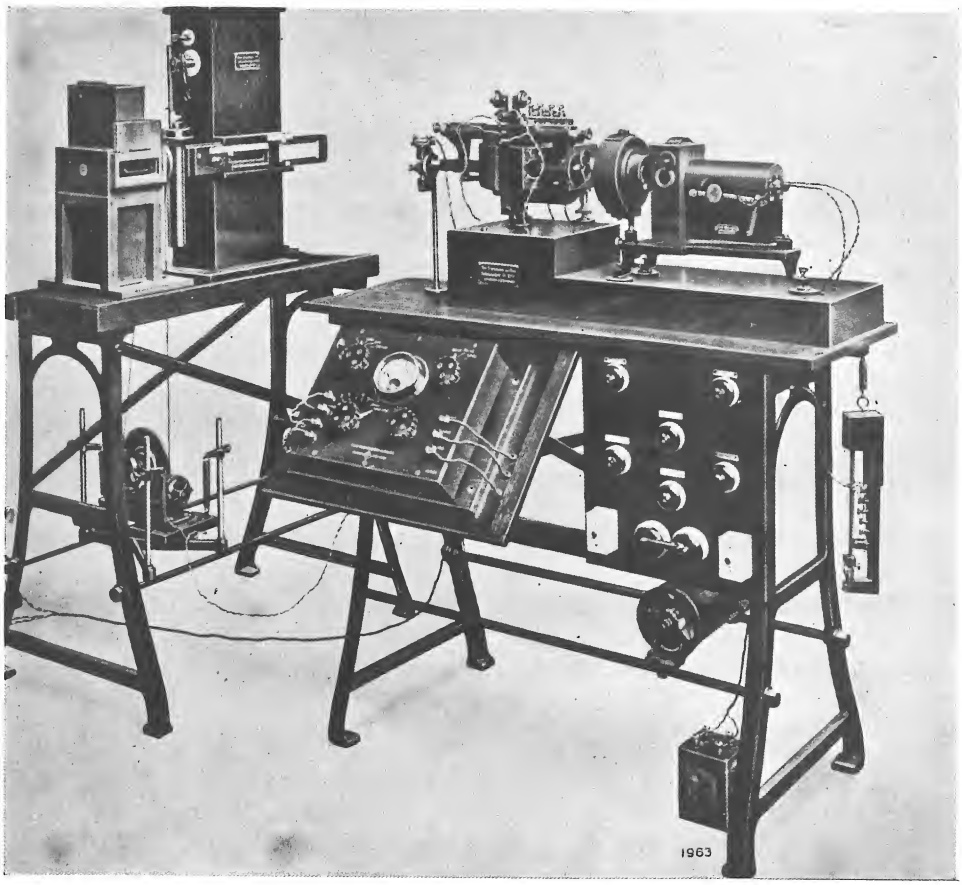


Figure 36. An English electrocardiograph of the nineteen-tens. On the left are photographic plate and paper cameras. (120, p.70).

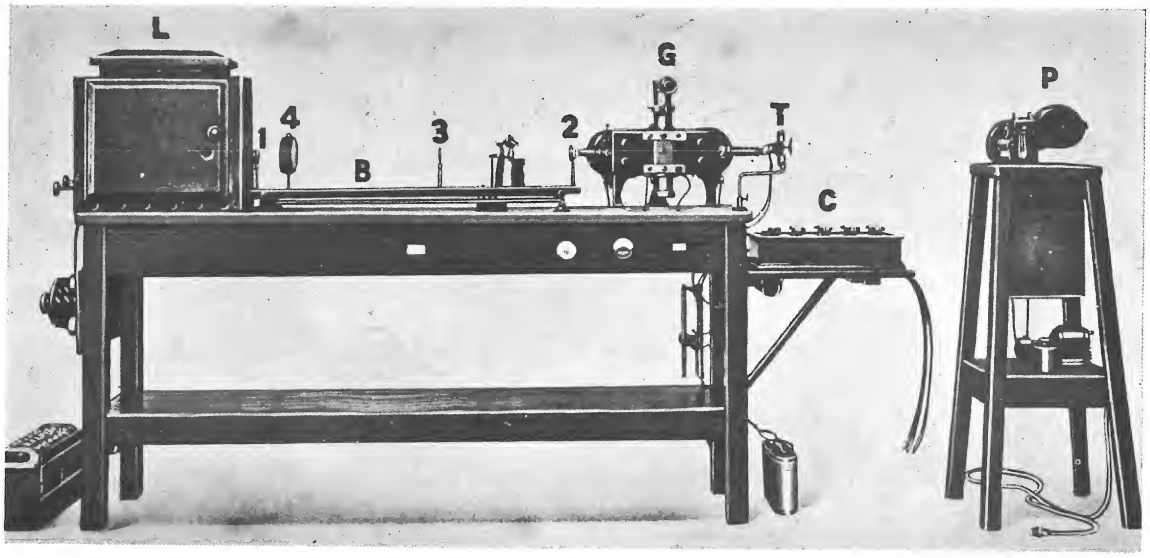
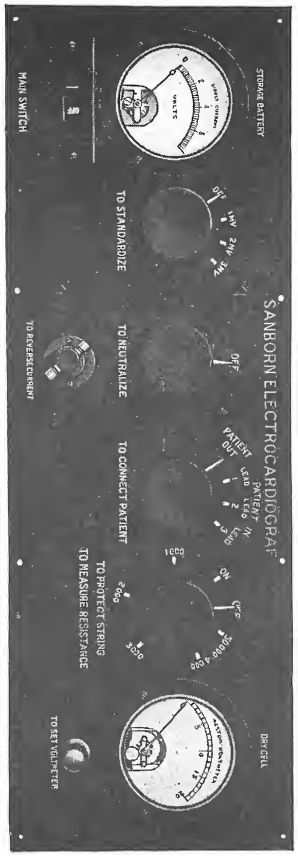


Figure 37. An American electrocardiograph of the nineteen-twenties. The light source L and string galvanometer G rest upon an optical bench. The ECG control box is mounted on the end of the bench, while the camera is on the right. (120, p.76).

Figures 36, 37, 38, 40 and 41 from Wiggers:
Principles and Practices of Electrocardiography,
St. Louis, 1929, The C.V. Mosby Company."



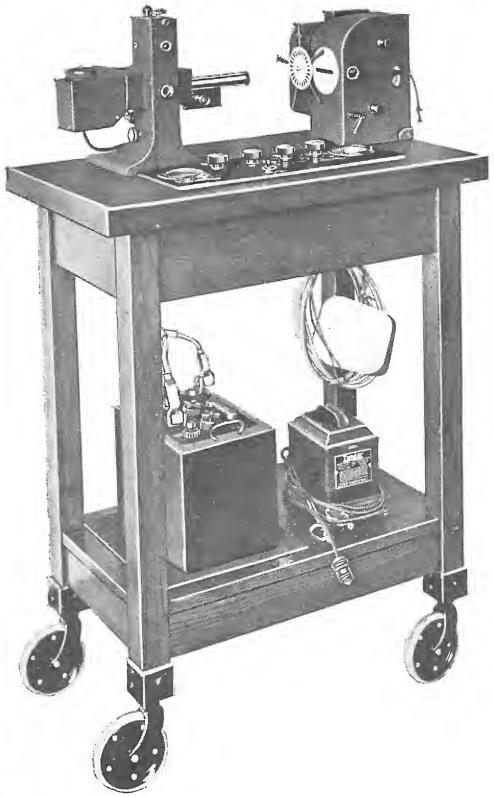


Figure 38. A Sanborn electrocardiograph of the early 1930's utilizing a string galvanometer. The storage battery powers the electromagnet, while the dry cell is used for standardization and compensation for the skin currents. (120, p. 90-91).

Figure 39. ECG electrodes of an earlier era. The outer crocks contain the leads immersed in zinc sulphate. The inner vessels are porous and contain saline and cotton wool to form a comfortable bath of porridge-like consistency. (77, p. 6).

Figure 40. The Victor Electrocardiograph, the first amplifier type of electrocardiograph of widespread use in the United States.

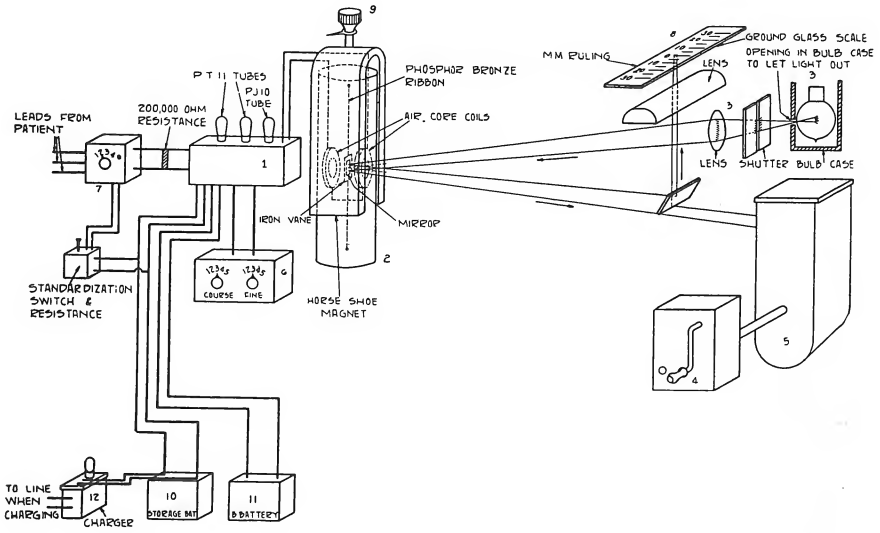


Figure 41. Schematic diagram of the Victor Electrocardiograph, illustrating battery operated amplifier, iron vane galvanometer, and wind-up phonograph motor driven camera.

the Duddell type. The first widely used commercial instrument of this kind in the United States was the Victor Electrocardiograph, modeled after an instrument developed by Mann in 1925 (83). It is illustrated in Figure 40 and is schematically diagramed in Figure 41 (120,p.92). Just as with the string galvanometer type of electrocardiograph, the record was produced by an optical system and moving photographic paper. The galvanometer utlized a small iron vane located in the field of a permanent magnet. Adjacent coils carrying the amplified signal current distorted the magnetic field, thereby causing the iron vane and attached mirror to twist. The oil bath method of Duddell was used to critically damp the galvanometer suspension.

Because of the extreme fragility of its quartz fiber, the string galvanometer was never particularly durable, even in so-called portable instruments. Due to the ruggedness of its galvanometer, the amplifier type of electrocardiograph rapidly gained acceptance. However, it was considered for some time an inferior type of instrument, even to the extent that some manufacturers would not sell an amplifier type electrocardiograph unless it was ancillary to a string galvanometer. In 1929 a comparison was made between the ECG records obtained from the same patients by the two different types of instrument. The conclusion was reached that "since the curves obtained from the amplifier-type instrument were essentially the same as those recorded by the string galvanometer, except for slight differences in amplitude, they [amplifier ECG's] may be considered satisfactory" (34,p.731).

During the 1930's the Sanborn Company introduced an electrocardiograph that was direct writing, i.e., that bypassed the photograpic process

and therefore gave an instant record. The recording paper is black and is covered with a thin film of white wax. A heated stylus lightly applied to the surface of the paper melts the wax and leaves a black on white impression. As the indicating pointer of a D'Arsonval type galvanometer, the heated stylus can trace out an ECG as the white wax paper moves by. Of course, the large galvanometer currents are obtained by amplification from the small surface potentials of the patient. Instruments of this type are illustrated in Figure 42 (22), which is a 1940 vintage and in Figure 43 (15) which is the modern version introduced in the 1950's.

More recent improvements have been the introduction of transistorized circuitry, which has lowered power consumption to the point that portable instruments are really practical. Although one may wonder if it is an improvement, there has also been the introduction of inkwriters, which after a long and unfortunate history, may have become reliable and mess free.

Vectorcardiography

Concepts

Vectorcardiography is the interpretation of ECG's from the view point that the surface potentials result from an electrical vector equivalent to the sum total of the heart's electrical activity. Most often this electrical vector is considered to be an electric dipole located at some fixed location within the body, and most probably in the region of the heart. Generally the tail of the vector is visualized as

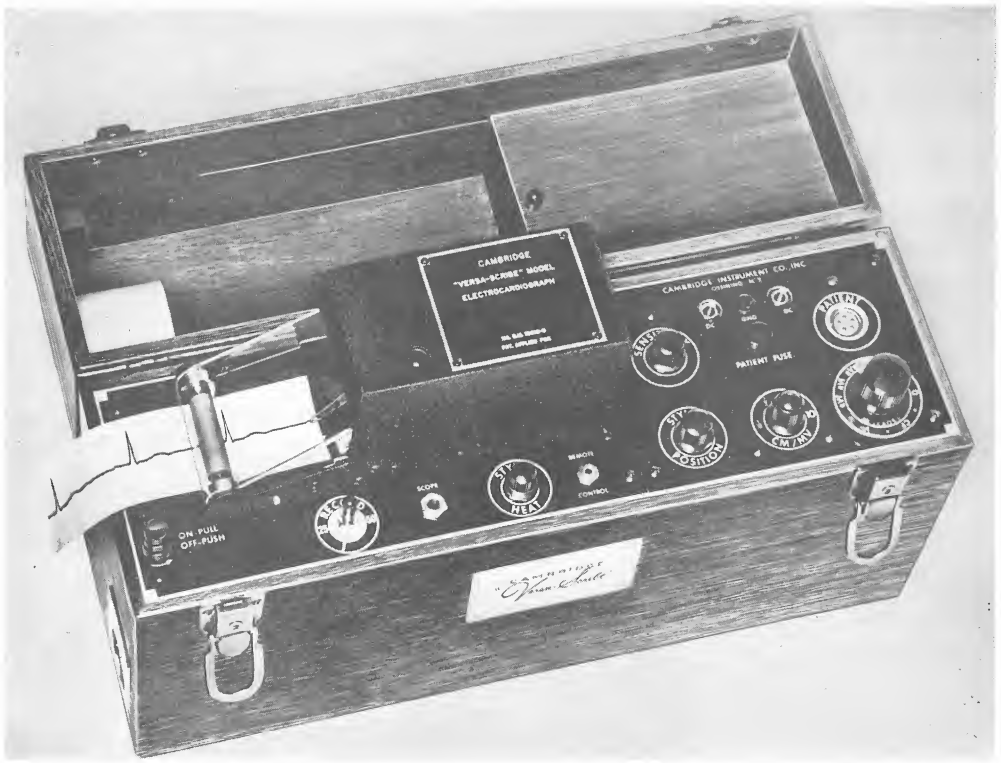


Figure 42. A typical electrocardiograph of the 1940's: heated stylus and packaged in a highly polished instrument box.

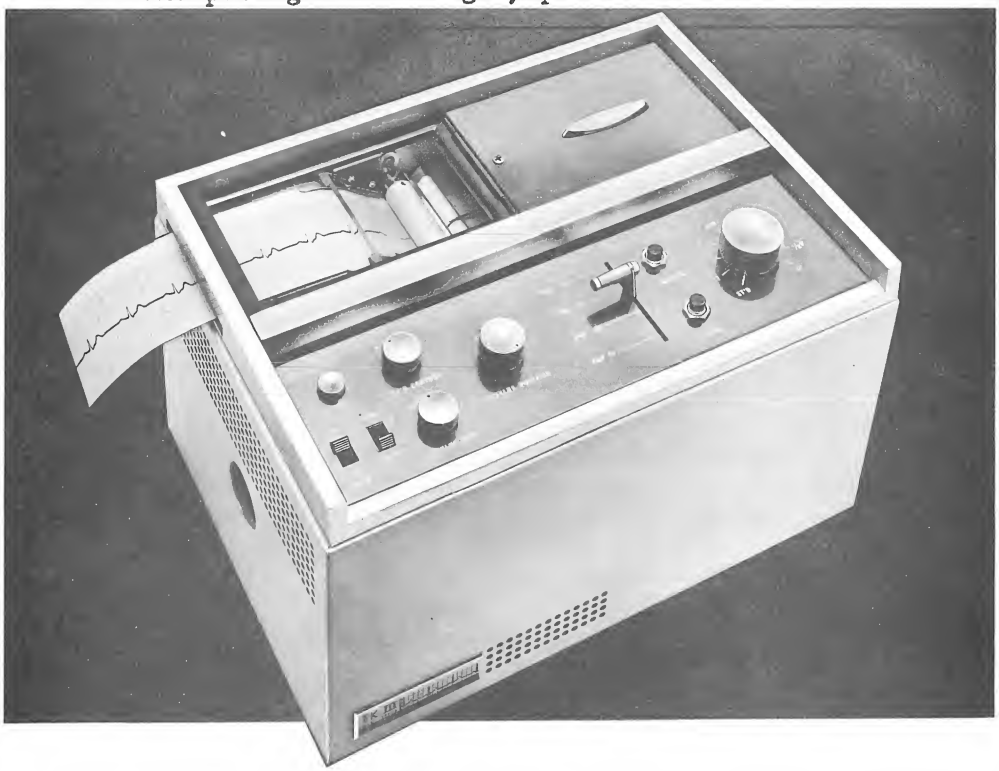


Figure 43. A typical electrocardiograph of the 1950's: heated stylus and packaged in a metal box with confectionary colors.

being fixed, while the head of the vector (or positive charge of the dipole) has the three translational degrees of freedom. Thus the electrical activity of the heart is equated with an equivalent electrical dipole whose magnitude and spatial orientation varies periodically with each heart beat.

This is by no means a new concept. It is implicit in Waller's 1889 illustration of the ECG field (Figure 24). Furthermore Einthoven's manifest potential is clearly formulated on a vector basis. His group was well aware of the spatial vector implications of the manifest potential, but they did not think it wise to introduce this complication into a field already complicated enough (113,p.547). In so far as vector presentation is technically difficult without the cathode-ray oscilloscope, this was probably a wise decision.

In 1920 Mann introduced the monocardiogram, whereby the three standard leads of the electrocardiogram were fused into a single curve, as is illustrated in Figure 44 (82,p.288). Since the potential of each lead varies with time, the manifest potential of which they are projections must also vary with time. Mann pointed out that it was relatively easy to plot these manifest potentials in rectangular coordinates by setting x equal to the potential of Lead I and setting y equal to the potential sum of Lead II and Lead III divided by radical three [(Lead II + Lead III)/$\sqrt{3}$]. The resultant x,y values can be plotted successively throughout a complete cardiac cycle and then can be connected by a smooth curve. The resultant curve he called the monocardiogram. As the x,y values are the rectangular coordinates of the manifest potential vector, it is seen that the monocardiogram is the projection of

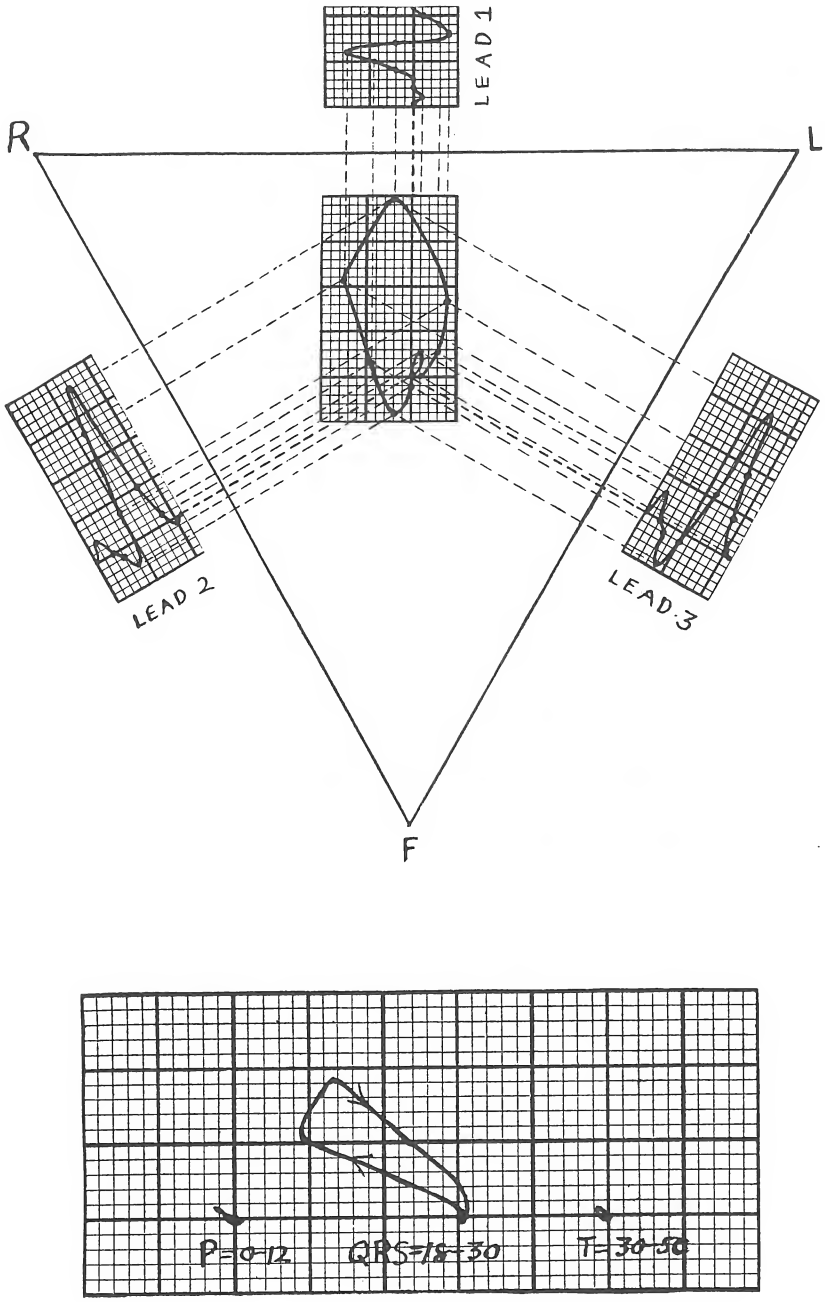


Figure 44. Mann's monocardiogram. The three leads of the electrocardiogram are fused into a single curve. In terms of the rectangular coordinates x and y: x = Lead I, while y = (Lead II + Lead III) $/\sqrt{3}$. Below are illustrations of the P loop, QRS loop, and the T loop. The numbers refer to timing in hundredths of a second.

the equivalent heart dipole on the plane defined by Einthoven's triangle. The lower half of Figure 44 illustrates the three loops of the monocardiogram generated in each cardiac cycle by the P, QRS and T waves. The tediousness of plotting the monocardiogram precluded its general use although Mann published a paper on its clinical use in 1931 (84). The concept was to lie virtually dormant for almost two decades until the general availability of the cathode-ray oscilloscope made the vector presentation a relatively easy matter.

Since only the standard leads were used, the monocardiogram gave the manifest potential vector projection on only the frontal plane. The spatial nature of the heart dipole makes it readily apparent that it would be possible to have projections on an infinite number of planes, but three planes would suffice to convey all spatial information. The orthogonal anatomical axes of the body are conventionally chosen to define these planes. Figure 45 (106,p.340) illustrates the orthogonal axes X,Y and Z and the three orthogonal planes: transverse, frontal and sagital. It is onto these planes that the periodic variations of the heart dipole is customarily projected in vectorcardiography.

In Figure 46 (113,p.586) the wire model illustrating the spatial wanderings of the dipole is a spatial vectorcardiogram. The shadows of this model projected on the walls are planar vectorcardiograms. Each shadow could also be projected onto two axes, then the variations along the axes are orthogonal electrocardiograms. In practice, one starts with what is hoped to be orthogonal electrocardiograms and constructs the planar vectorcardiogram, either by hand, or electronically with an oscilloscope. To obtain a spatial vectorcardiogram one either mentally

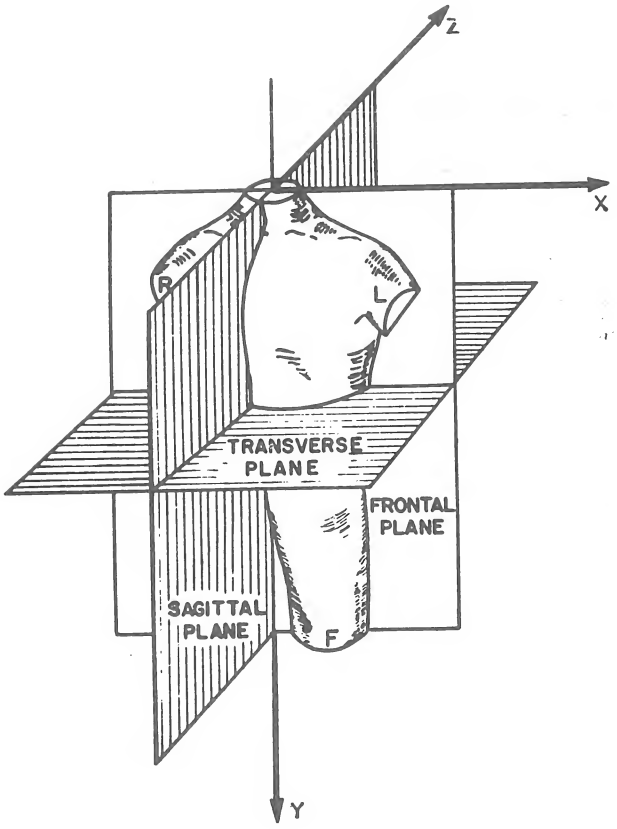


Figure 45. Orthogonal axes and planes defined for vectorcardiography. X, Y and Z are the axes along which bipolar electrocardiograms are taken. Any two ECG's plotted against each other will define a planar vector in the transverse (XY), frontal (XY), or sagittal (YZ) plane. Simultaneous inclusion of the third ECG will define a spatial vectorcardiogram.

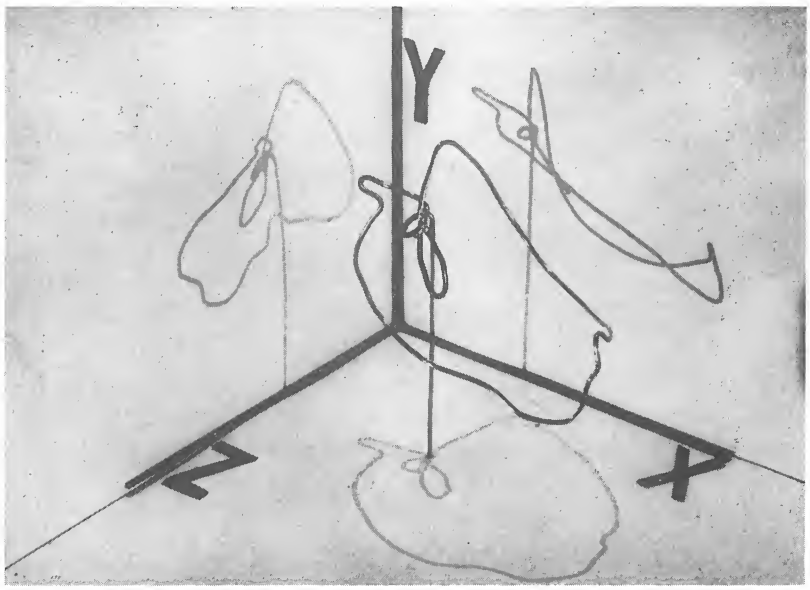


Figure 46. Wire model of a spatial vectorcardiogram. The shadows are planar vectorcardiograms. Variations along axes are ECG's.

integrates the three planar vectorcardiograms, builds a wire model, or stereoscopically views two planar vectorcardiograms which are rotated ten or twenty degrees from each other (either mathematically, or electronically).

To obtain orthogonal electrocardiograms is no simple matter. Three commonly used systems for vectorcardiography are illustrated in Figure 47 (106,p.341), of which the two cube systems are conceptually orthogonal as regards electrode placement. However it must be noted that electrocardiograms taken along these cubic axes are bipolar, and as such electrode placement has a critical effect upon the ECG record. The result is that these X,Y, and Z ECG's are not electrically orthogonal, i.e., they are not mutually consistent with an orthogonal projection from the same manifest potential vector. The third system illustrated is Wilson's tetrahedron and its leads are obviously neither anatomically nor electrically orthogonal.

Four orthogonal systems which are considered to be more accurate than the cube or tetrahedron systems (74) have been devised (36,52,90, 113). The most popular of these is the Frank system which is illustrated in Figure 48 (36,p.738). This system combines a high degree of orthogonality with the convenience of a minimal number of electrodes (seven). Critically placed electrodes and a computing resitive matrix compensate for torso inhomogeities in attempt to achieve isotropy as well as orthogonality of projection for the equivalent heart dipole.

Theoretical Bases

The dipole hypothesis is open to justifiable criticism on both

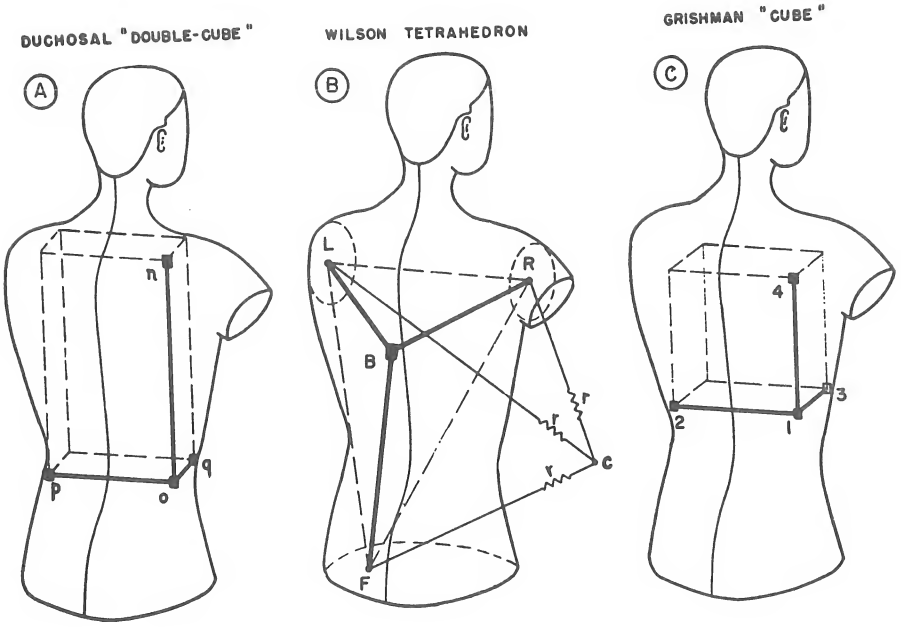
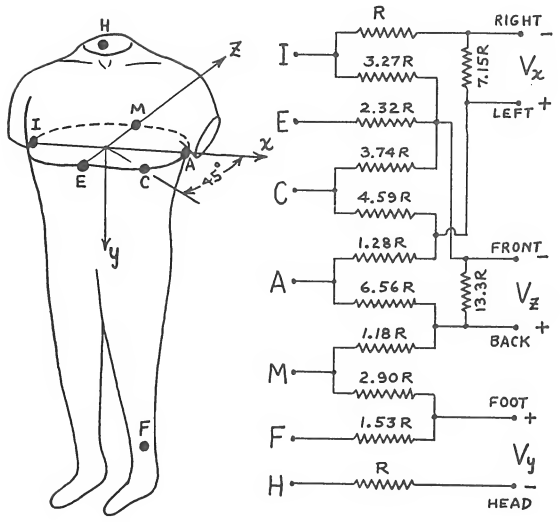


Figure 47. Three commonly used systems for vectorcardiography. Systems A and C are bipolar and attempt to be orthogonal. System B uses the central terminal C and is therefore unipolar. It is based upon a tetrahedron and is clearly not orthogonal.



"By permission of the American Heart Association, Inc."

Figure 48. The Frank system for spatial vectorcardiography. Using a minimal number of electrodes this system attempts to achieve isotropy as well as orthogonality. Critically placed electrodes and a resistive matrix compensate for torso inhomogeneities. Accuracy of angle is ± 5%, while length is ± 20%.

physiological and electrical grounds. The many millions of cardiac muscle fibers are located throughout the heart volume and are asynchronously activated. This results in many dipoles, residing in many places, being activated at many different times. The surface potential effects of these fibers can be partially ascribed to an equivalent dipole, but it is not reasonable to expect that the dipole's location, any-more-so than its magnitude or orientation, is fixed. Although, of course, one would expect the location to be somewhere in the region of the heart. A full description of the potential variations at the surface would require the addition of higher order multipoles (quadrupole, octupole, etc.) that would also have to vary, with time, in location, magnitude and orientation. The experimental technique for the investigation of multipoles is extremely difficult, and therefore to what extent these higher order poles contribute to the ECG's of normal and pathological subjects is yet to be determined (97).

In a critical review of vectorcardiography Okada points out that there have been several series of experiments on normal subjects that appeared to verify the dipole hypothesis to a high degree of accuracy (97). Unfortunately these experiments tested the necessary conditions for the applicability of the dipole hypothesis, which is not the same as those conditions sufficient to validate the theory. Furthermore, the experimental procedures were complex and tedious, the instrumentation did not seem adequate, and the results were sometimes difficult to evaluate. In addition, there were several instances where the dipole hypothesis was invalid. Okada feels that the single equivalent dipole generator is not accurate enough to justify the exclusive use of

vectorcardiography for diagnosis, a modus operandi that no reputable cardiologist would subscribe to anyway. However, he did conclude that (97,p.98): "there is absolutely no conflict between the theoretical limitations outlined in this [his] paper and the clinical usefulness of empirical vectorcardiography. ... The main purpose of this [his] paper is not to discourage empirical vectorcardiography, but rather to point out the theoretical limitations which should ... help limit interpretations of clinical and experimental results within the confines of known physical and mathematical limitations."

Thus there is the suspicion that the central hypothesis of vectorcardiography is on somewhat shaky theoretical ground. This should not overly disturb us, because it is not at all uncommon in newly emerging theories. Many great strides have been made in the past on the basis of unjustified assumptions about an oversimplified model. Today the call is for better instrumentation and more exhaustive studies so that the limits of the present hypothesis can be precisely ascertained, and so that better hypotheses can be proposed in the future.

Instrumentation

At the same time that Mann was developing the concept of the vectorcardiogram (1920), a major technololgical advancement was taking place that was to ultimately revolutionize the presentation of data. At the Western Electric Company J. B. Johnson was developing the hot cathode Braun tube, which could form a usable electron beam with an anode voltage of several hundred volts instead of the several tens of thousands of volts previously required (69). Consequently the electron beam

acceleration was much less, and this resulted in lower beam velocities in the region of the deflection plates. Therefore the beam was not as "stiff" and could be deflected easily by relatively low potential differences between the deflection plates.

In 1922 Gasser and Erlanger constructed the first cathode-ray oscilloscope using Johnson's tube. Figure 49 (41,p.502) is the schematic diagram of this original instrument, which interestingly had a sensitivity of 4 mV/cm. They used the oscilloscope to study the action currents of nerve in response to artificial electrical stimulation. For this work in 1944 they were awarded the Nobel prize in physiology and medicine.

In 1925 Dock described the use of the cathode-ray oscilloscope for electrocardiography (23). He developed a small (refer to Figures 36 and 37 for large) portable electrocardiograph to be used in conjunction with an x-ray machine. "With no string or delicate part to be damaged by the currents of the x-ray unit, the cathode-ray oscillograph ... has proved quite satisfactory for making electrocardiographs." His instrument had a sensitivity of 1 mV/cm, and utilized a moving film camera to obtain a continuous record. The film was applied directly to the face of the cathode-ray tube and moved at speeds up to 15 cm/sec. The electrocardiographic leads were ac coupled to the grid and directly connected to the cathode of the input stage. With the components used this resulted in a low frequency response down to .1 cycle per second.

"The first practical, commercial cathode-ray oscillograph was introduced in the United States by Allen B. DuMont Laboratories, Inc. in 1932" (1,p.86). The usefulness (or the novelty) of the instrument soon created a public appetite. By 1935 there were several different types

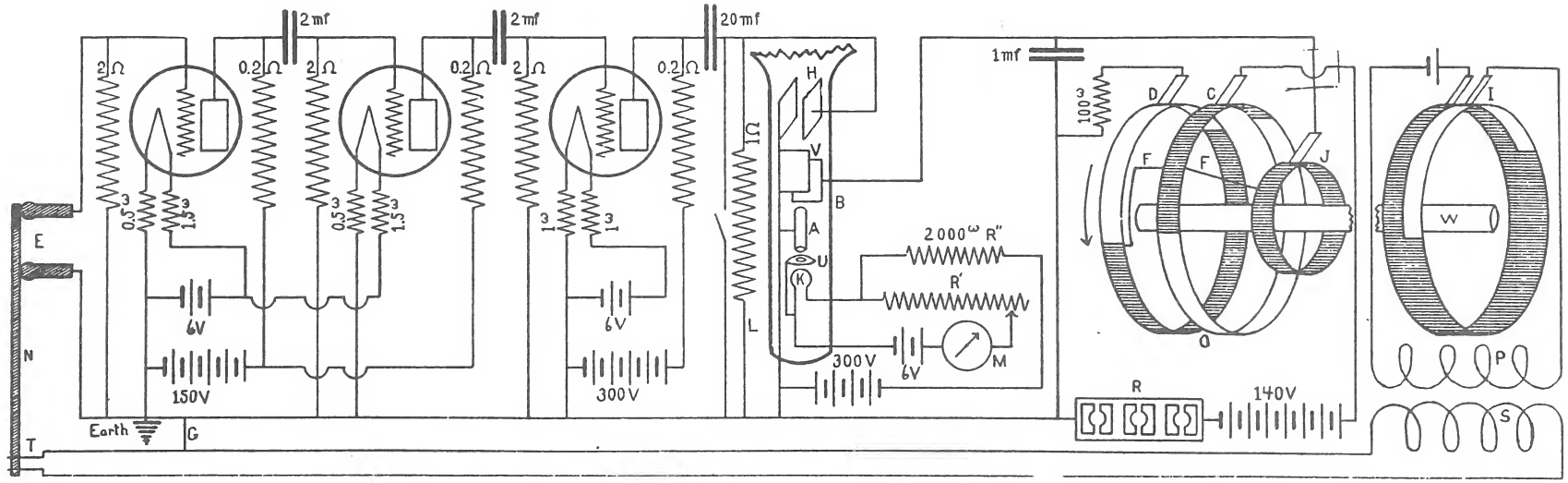


Figure 49. Schematic diagram of the first oscilloscope, built by Gasser and Erlanger in 1922. This scope used the hot cathode version of the Braun tube developed by J. B. Johnson at Western Electric. Their figure legend reads: "Diagram of Braun tube oscillograph arranged for observation of nerve action currents. N = nerve, killed end shaded. E = non-polarizable electrodes connected through the three-stage amplifier with the horizontal plates, H, [defines plate orientation and not beam deflection], of the Braun tube, B. K = Wehnelt cathode; A = tubular anode; U = diaphragm; L = leak on horizontal plates; M = ammeter; R' = regulating resistance in filament circuit, K; R" = resistance in anode circuit. The vertical plates, V, [defines plate orientation and not beam deflection], connect with the spreading device [sweep], O. D = discharging wheel, C = charging wheel and J = continuous contact wheel (metallic parts shaded) all connected conductively by F; R = charging resistance to 1 mf. condenser. I = rotating interupter in primary circuit, P; S = secondary coil; T = stimulating electrodes grounded through G. 1Ω = 1 megohm. For the intertube leaks labeled 2Ω, 0.5Ω was used."

of oscilloscopes and over a dozen American manufacturers (103) including some well known as well as some nostalgic names: National Union, RCA, Kaltman-Romander, National, Clough Brengle, General Radio, United Sound, Dayrad, Supreme, Hickok, Triumph. Presumably there were a like number of European manufacturers as well.

Shortly after the availability of commercial oscilloscopes the successful use of the cathode-ray tube for the automatic recording of electrocardiographic vector loops was independently and almost simultaneously announced by Schellong in 1936 (107), and in 1937 by Hollman and Hollman (63), Wilson, Johnston and Barker (125), and Sulzer and Duchosal (115). As might be expected there were also many descriptive names applied to the cardiac vector loops: planelectrocardiogramme (115), planogramme (116), triogramm (62), vektordiagramm (108). In 1938 Wilson and Johnston granted the priority of the name "monocardiogram" (82), but suggested that "vectorcardiogram" was more descriptive of the true nature of the vector loops.

The dynamic presentation of the electrocardiogram in two dimensions was only made possible by the cathode-ray tube (CRT) display. To anyone who observes the vectorcardiogram being traced out on a CRT the vector nature of the equivalent heart dipole is dramatically brought to mind. It is not surprising that before long there was the desire to dynamically display this dipole in the three dimensions of space. However, spatial vectorcardiography requires stereopsis, or depth perception, and to truly do this requires three dimensions. This can be attained through 3D wire models (5,113), but model building is hardly a dynamic presentation.

The perception of depth depends upon many inputs to our central

nervous system: the primary (physiological) cues and the secondary (learned) cues. The primary cues are the actual physiological inputs such as the accomodation of the eye lens, the convergence of the eyes' optical axes, and the retinal disparity of the images received by the two eyes. Secondary cues are those learned things that a painter might use to create the illusion of depth: linear perspective, aerial perspective, relative size, interposition, gradients of texture, patterns of light and shadow, and relative motion (if possible).

A very satisfactory illusion of depth can be had by tricking the physiological cue of retinal disparity. By providing each eye with a flat, but slightly different (rotated), view of the same object the mind interprets the two views as retinal disparity and therefore fuses them into a single 3D object. To obtain these two slightly different views in 1944 Vastesaeger and Rochet placed electrodes on the shoulders in such a manner as to obtain two different electrical plane projections tilted out of the frontal plane (117). In 1950 Cronvich produced a similar stereoscopic image by rotation out of the frontal plane with an electrical network rather than by extra electrodes (13,p.605).

The use of the cathode-ray oscilloscope for the presentation of three dimensional data, either in projection or in 3D, was discussed at some length by Schmitt in 1947 (112). Following up on some of Schmitt's suggestions, Milnor, Talbot and Newman constructed the panoramic vectorcardiograph, which transformed the incoming orthogonal x,y,z signals into orthogonal x',y',z' signals which were equivalent to a rotation of the spatial vectorcardiogram (91). Thus they were able to obtain planar vectorcardiograms as a projections of the spatial vectorcardiogram from

any angle.

A different type of vectorcardiograph was described by Briller, Marchand and Kossman in 1950 (10). Their interest was not so much the spatial orientation of the vector loops, but rather the ability to investigate each vector loop (P, QRS, T) individually and without interference (overlap) from the other loops. Unfortunately they named their instrument the differential vectorcardiograph because of its ability to look at different parts of the ECG. The present author feels that a word such as "selectable" would be just as descriptive and would not be subject to misunderstanding. They obtained a "differential" display by unblanking the CRT only during the time period of the individual loop under investigation. The unblanking period was selected by electronic delay circuits that were triggered off of the leading edge of the P wave. Using a dual beam CRT they obtained frontal and sagittal projections which were also Z-axis modulated by a timing wheel and photocell.

In 1954 Hellerstein, Shaw and Sano described an "electrical dissector" which could be used with any commercially available dual-beam oscilloscope (51). In essence the electrical dissector was a self-contained Z-axis modulator combining a time mark generator and selectable unblanking. The delay circuits were triggered by the QRS complex obtainable from any ECG amplifier.

In 1962 Isaacs carried the commercialization one step further by describing how utilization of Tektronix 160 Series waveform and pulse generators and some simple modifications to a Tektronix Model 502 Dual-Beam Oscilloscope could accomplish the electrical dissection and

CRT display at a considerable saving in time and cost (67). However, this instrument was able to display only one vectorcardiogram at a time but by turning a switch the two scaler leads (x and y, y and z, or x and z) composing the vector loop could be displayed on any convenient time scale. Isaacs and his associates subsequently assembled a system* utilizing Tektronix instruments that simultaneously displays (on two oscilloscopes) calibration signals, the three orthogonal scaler leads (x,y,z), and the three orthogonal vectorcardiograms synthesized from them. The heart of this system is two Tektronix Type 3A74 Fourtrace A Amplifiers which by synchronization can display four simultaneous X-Y plots on the same single-beam CRT. The Vector Electrocardioscope System to be described in the next section is a refinement of and extension upon Isaac's system.

In the 1950's the surge of interest in vectorcardiography was such (3,5,10,13,36,37,44,51,52,71,74,88,89,90,91,100,105,111,113,114) that manufacturers began designing instruments specifically for vectorcardiography. There is on the market a complete "vectorscope" which integrates into one instrument the patient leads, lead switching, ECG amplifiers, and the oscilloscope display (48).There are several other instruments (68,96,104) which, incorporating only the patient leads, lead switching,and ECG amplifiers in a "vectoramplifier," require an auxiliary oscilloscope for display. It is anticipated that in the next decade there will be considerably more vectorcardiographic instrumentation

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* "Improved Instrumentation for vectorcardiography," manuscript in preparation for publication.

available, since it is universally recognized that "the automatized techniques for biological use are at present lagging behind the techniques in physical and chemical research and their industrial application" (132,p.171).

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